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CLINICAL PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN
THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK, ETC.

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TO MY FRIEND

AND

MASTER IN THE CLINICAL ART,

PROFESSOR WILLIAM H. DRAPER, M.D,,

THIS VOLUME IS AFFECTIONATELY INSCRIBED.

E. C. SEGUIN.

PREFACE.

AN apparently unlimited interruption having taken place in my professional life, the idea occurred of reprinting my various medical contributions for private circulation.

Yielding to the advice of several friends, I have consented to the issue of a larger edition for regular sale.

In offering these articles, etc., to the profession I need make no apology, because I do it in the same spirit that led me to publish them separately. I have, I hope, a due appreciation of the faults in some of the essays, of the shortcomings of many others, and of the fact that some of them possess only an historical interest. The redeeming feature in the collection will perhaps be the series of observed facts, faithfully recorded, which may prove of some use to the practical physician as well as to the pathologist.

Without an editor this volume could not have appeared. I desire to thank my friend Dr. Amidon for the patience and accuracy he has shown in the performance of his task, which involved among other things the reduction of all measures to the metric and centigrade scales, and the verification of every reference.

E. C. SEGUIN.

ZURICH, SWITZERLAND, *August*, 1883.

CONTENTS.

PAGE
THE Use of the Thermometer in Clinical Medicine.......... 1
On the Subcutaneous Use of Sulphate of Quinine in Cases of Malarial Neuralgia.......... 10
On Treatment of Malarial Fevers by the Subcutaneous Use of the Sulphate of Quinine.......... 15
The Hypodermic Injection of Quinine.......... 22
A Statement of the Aphasia Question, together with a Report of Fifty Cases... 27
    Cases in which Aphasia occurred with Right Hemiplegia.......... 44
    Cases in which Aphasia occurred with Left Hemiplegia.......... 52
    Cases in which Aphasia occurred with Double Hemiplegia.......... 52
    Cases in which Aphasia occurred without Hemiplegia.......... 53
Case of Trichinosis.......... 55
A Case of Acute Ocular Œdema—cause unknown.......... 56
Cas de Lesion probable de la moitié latérale droite de la moelle épinière, dans la région cervicale inférieure, ayant produit de l'anesthésie d'un côté et de la paralysie de l'autre.......... 57
Autopsy of a Case of Mania.......... 62
Contributions to the Pathological Anatomy of the Nervous System.......... 72
Case of General Paresis of the Insane.......... 76
Infantile Spinal Paralysis.......... 84
A Case of Traumatic Brachial Neuralgia treated by Excision of the Cords which go to form the Brachial Plexus,.......... 97
On the Inhibitory Arrest of the Act of Sneezing, and Its Therapeutical Applications.......... 118
Description of a Peculiar Paraplegiform Affection. (Tetanoid Paraplegia.).. 127
Lecture upon the General Therapeutics of the Nervous System.......... 139
An Outline of the Physiology of the Nervous System.......... 164
On Hysterical Symptoms in Organic Nervous Affections.......... 180
Syphilitic and Simple Pachymeningitis.......... 195
A Clinical Contribution to the Study of Post-Paralytic Chorea.......... 197
Contribution to the Study of Localized Cerebral Lesions.......... 202
    Cases in which a more or less limited Cerebral Lesion produced Aphasia. 202
    Cases in which a limited Cerebral Lesion caused Paralysis.......... 214
    Cases in which localized Cerebral Lesions gave rise to Localized Convulsions or Spasm.......... 215
The Abuse and Use of Bromides.......... 226
A Contribution to the Therapeutics of Migraine.......... 242
Bulbar Paralysis. (Atypical Case of Labio-Glosso Pharyngeal Paralysis)..... 249

PAGE
Localized Basal Meningitis in Children........ 251
A Clinical and Therapeutical Contribution to our Knowledge of Cervical Paraplegia........ 256
A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis........ 264
A Clinical Lecture on Syphilitic Cerebral Lesions........ 276
Lectures on the Localisation of Spinal and Cerebral Diseases........ 283
Lecture I.—Summary. Introductory Remarks........ 283
Historical Considerations........ 287
Lecture II.—Synopsis: Systematic Lesions of the Spinal Cord: Lesions in the Æsthesodic System: Lesions in the Kinesodic System........ 293
Lecture III.—1. Systematic Diseases of the Spinal Cord continued: Lesions in the Kinesodic System........ 300
2. Non-Systematic or Focal Lesions of the Spinal Cord: Lesions at different heights in the Organ........ 304
Lecture IV.—Non-Systematic or Focal Lesions of the Spinal Cord continued: Lesions involving one lateral half of the Spinal Cord, in its Lower and Upper Regions: Diagnosis of Spinal Hemiplegia........ 309
Anatomy and Diseases of the Medulla Oblongata........ 311
Lecture V.—Summary........ 318
Lesions of the Basis Cerebri........ 318
Physiological Anatomy........ 319
Symptoms of Lesions of the Basis Cerebri........ 322
Lesions of the Pons Varolii........ 322
Lesions of the Crura Cerebri........ 324
Lesions situated at the Base of the Brain anteriorly to the Crura........ [illegible]
Lecture VI.—Summary........ [illegible]
Lesions of the Great Ganglia at the Base of the Brain, and of the white substance of the Hemispheres........ [illegible]
Lesions of the Basal Organs........ [illegible]
Lesions of the Internal Capsule........ [illegible]
Lesions of the Cerebellum........ [illegible]
Lecture VII.—Summary: Anatomy and Lesions of the Cortex of the Brain: the Chief Cortical Motor Centres and Broca's Speech-centre: Localised Lesions of the Cortex Cerebri: Diffused Lesions of the same........ [illegible]
Lecture VIII.—Surgical Aspects of the question of Cerebral Localisation: Cranio-cerebral Topography and its Utilisation in Diagnosis and in Operative Procedures........ [illegible]
The Diagnosis of Progressive Locomotor Ataxia........ [illegible]
Report on Aconitia in the Treatment of Trigeminal Neuralgia........ [illegible]
A Contribution to the Medicinal Treatment of [illegible] Neuralgia........ [illegible]
Dermatitis Produced by Three Preparations of Iodine in the Same Subject........ [illegible]
Myelitis of the Anterior Horns of Traumatic Origin........ [illegible]
The Present Aspect of the Question of Traumatic [illegible]........ [illegible]
The Use of the Actual Cautery in Medicine........ [illegible]
Traumatic [illegible] of One Year's Standing [illegible]........ [illegible]
Case of [illegible]........ [illegible]

PAGE
"Folie À Deux" 405
Case of Hemiplegia with First Symptoms in Foot, and a Limited Cortical Lesion 407
Case of Slow Pulse and Epileptiform Convulsions 409
A Contribution to the Pathology of Acute Central Myelitis 412
Paraplegia in Syphilitic Subjects 413
A Case of Movable Kidneys; Remarkable Voluntary Control Over These Organs 415
Case of Cerebral Hemorrhagic Pachymeningitis 416
The Intra-Buccal Method of Faradizing the Lower Facial Muscles 419
On the Coincidence of Optic Neuritis and Subacute Transverse Myelitis 421
A Case of Mysophobia 429
On Occipital Headache as a Sympton of Uræmia 432
The Localization of Diseases in the Spinal Cord 436
A Case of Abscess of the Left Frontal Lobe of the Cerebrum, with Special Reference to Localization 452
On the Early Diagnosis of Some Organic Diseases of the Nervous System 457
Report on the Use of Hyoscyamia as an Hypnotic and Depresso-Motor 472
    Historical 472
    Clinical Experience with Hyoscyamia as an Hypnotic 477
    Cases Reported by Dr. A. B. Ball 480
    A Case of Delirum Tremens, by Dr. F. P. Kinnicutt 482
    A Case Contributed by Dr. Andrew H. Smith 482
    Case of Morbid Dreams, by Dr. E. C. Seguin 482
Hyoscyamia as a Depresso-Motor 484
    do in Choreiform Affections 488
The Physiological Effect of Aconitia in Posterior Spinal Sclerosis: Can It Become an Aid in Differential Diagnosis? 492
A Case of Diphtheritic Ataxia and Paralysis from Anal Diphtheria—Cure 493
A Second Contribution to the Study of Localized Cerebral Lesions 495
    Expressions of Symbols, by Dr. A. B. Ball 511
    Comprehension of Symbols, do do. 514
    Dr. Seguin's Report of the Autopsy 518
    Concluding Observations by Dr. Seguin 520
On the Use of a Feebly Alkaline Water as a Vehicle for the Administration of the Iodide and Bromide of Potassium, etc 529
History of Attempts Made to Cure Three Cases of Chronic Trigeminal Neuralgia 534
Importance of the Early Recognition of Epilepsy 540
On the Methods of Diagnosis in Diseases of the Nervous System 558
The Efficacy of Iodide of Potassium in Non-Syphilitic Organic Diseases of the Central Nervous System 579
    Ideas of Different Authors bearing on the Subject 592
On the Efficient Dosage of Certain Remedies Used in the Treatment of Nervous Diseases. (I.) 594
On the Efficient Dosage of Certain Remedies Used in the Treatment of Nervous Diseases. (II.) 599
    Crystallized Aconitia of Duquesnel 599
    Phosphorus and Phosphide of Zinc 602
    Crystallized Nitrate of Silver 605
Case of Injury to the Motor Area of the Brain, with Exhibition of the Patient. 608

PAGE
Remarks on the Frequency of Headache and Choked Disc with Tumor of the Brain........ 609
Vertebral Cancer and Paraplegia........ 612
Myelitis Following Acute Arsenical Poisoning by Paris or Schweinfurth Green........ 616
On a Peculiar Cutaneous Lesion (Ulcus Elevatum) Occurring During the Use of Bromide of Potassium........ 629
A Case Illustrating the Coincidence of Diseases: Cervico-Brachial Neuralgia and Aneurism of the Innominate Artery........ 636
Aneurism of the Cœliac Axis........ 639
Hysterical Convulsions and Hemi-Anæsthesia in an Adult Male: Cured by Metallo-Therapy (Gold)........ 641
Two Cases of Glycosuria: One True and One Simulated........ 644
Note on Cranio-Cerebral Topography. (Illustrated)........ 647
The Treatment of Mild Cases of Melancholia at home........ 655
The Cultivation of Specialties in Medicine........ 672

OPERA MINORA.

THE USE OF THE THERMOMETER IN CLINICAL MEDICINE.*

BELIEVING that the matter may prove of interest, and that the attention of practitioners may thus be called to a means of diagnosis and prognosis not second in importance to any single one hitherto employed, the following cases of pneumonia, observed and treated in the New York Hospital during the month of January, 1866, are given as illustrative of the application of thermometry in disease; together with an abstract of the highly interesting and elaborate paper of Dr. L. Thomas, of Leipzig, on the thermal phenomena of pneumonia. ("Ueber die Temperatur Verhältnisse bei croupöser Pneumonie," *Archiv der Heilkunde;* Bd. V, S. 30–36.)

The cases are accompanied by a diagram, *fac-simile* of the tables of "Vital Signs" used at the bedside to make the daily record of temperature, pulse-beats and respirations. This one only differs from ours in that on it are represented the curves for three cases, whereas usually but one case is put upon a table. No further explanation of the diagram is necessary, except to state that the heat is registered in decimal parts of degrees; the pulse and respirations, of course, by whole numbers.

---

* From the *Chicago Medical Journal*, May, 1866.—This article and the observations leading to it form the starting point of Medical Thermometry in the United States.

The annexed table for recording vital signs was designed by Dr. William H. Draper and Dr. Seguin conjointly. At the time Dr. Seguin was senior assistant in the medical department of the Hospital, and during the remainder of his service (until Aug. 1, 1867) gave a great deal of his time to the thorough practice of thermometry. It then became part of the hospital routine, and has so continued. It may be safely claimed that medical thermometry spread from this institution as a focus.—R. W. A.

Two observations are found in each daily column, one in the morning made at 9.10 A.M., one for the latter part of the day made at 4.15 P.M.

For want of space the cases are much abbreviated; the physical examinations being nearly omitted. This may be justified by stating that the diagnosis in each case was verified by the attending physician, Dr. Wm. H. Draper, and that careful daily examinations were made by Dr. J. Haven Emerson, the talented house physician of the Hospital.

CASE I.—G., æt. 28. Steamship fireman.

Admitted Jan. 11th, 1866. Was taken ill on 6th with chill, pain in right side, etc.

On admission, considerable febrile disturbance of rather low type; tongue much coated, inclined to dry in middle; general condition fair. Physical examination reveals dullness, bronchial breathing and voice, and crepitant râle over limits of right upper lobe; most marked in supra-spinous fossa. Expectoration characteristic. Is ordered decubitus, an oiled-silk jacket, at night a Dover's powder, and

℞. Pulv. ipecac., .12
Liq. ammon. acetat., 120.
M. Cap. one tablespoonful q. 4. h.

Jan. 12. Consolidation progressing; expectoration thin, containing some pure blood; had epistaxis yesterday; tongue rather dryish and brown coated. Ordered, Sherry wine, 180 cc., to be taken with milk; continue mixture.

Jan. 15. Defervescence occurred during last twenty-four hours, with a fall of 3.5° C. (See table.) General bronchitis has supervened, and masks the physical signs in the affected lobe. Tongue is quite moist and cleaner. Stop mixt.

Jan. 17. Quite convalescent. Ordered, continue wine and take quinine sulph., .12 t. i. d.

Jan. 27. For a week has sat up and been about. Bronchitis is gone, and he is discharged cured.

CASE II.—R. S., æt. 26. Seaman.

Admitted Jan. 16th, 1866, having been ill four days. Sickness began with chill, pain in right side, fever and cough.

On admission he presents all the objective signs of pneumonia, involving the right lower lobe, in stage of red hepatization; expectoration rust-colored; general condition good. Ordered, decubitus, and at night 1 c. c. of Squibb's liq. opii co., and the ipecac. mixture, as given above, every four hours.

An oiled silk jacket, lined with cotton, was applied over the chest.

Jan. 21. A small fall in the evening temperature indicates the limitation of the disease. A pleuritic friction sound is heard at the angle of the scapula. Cont. treatment.

Jan. 23. Yesterday complete defervescence did not occur, because the inflammation extended to the upper lobe of same side; tympanitic dullness is found over the angle of the scapula. Defervescence did occur this A.M. Yesterday, patient had a little epistaxis and diarrhœa, which latter was checked by suppos. opii, (.12) p. r. n. Expectoration less colored, more abundant and muco-purulent. Cont. treatment.

Jan. 24. Vital signs down to normal standard.

Jan. 31. Resolution took place rapidly; patient has been up for a few days. Some dullness remains at apex and over lower lobe, and at the angle of the scapula are still heard pleuritic friction sounds, some subcrepitant râles and broncho-vesicular breathing.

Feb. 5. Patient is discharged, cured.

CASE III.—S., æt. 19. Coal-passer on steamship.

Admitted Jan. 25th, 1866. Illness began on 21st with chill, pain at lower ribs of right side, cough and fever.

On admission there is much pain, great febrile movement and dyspnœa; expectoration rust-colored, very characteristic; patient presents all the physical signs of inflammation involving the right upper lobe. General condition excellent, except previous malarial poisoning. Ordered, an oiled-silk jacket, a hypodermic injection of morphia (.60 of Magendie's sol.) and the mixture used in the cases above, in the same doses. Decubitus.

Jan. 27. There is complete hepatization of affected lobe. Defervescence occurred during last twelve hours, with a fall of 3.7° C. Had epistaxis yesterday. Continue treatment.

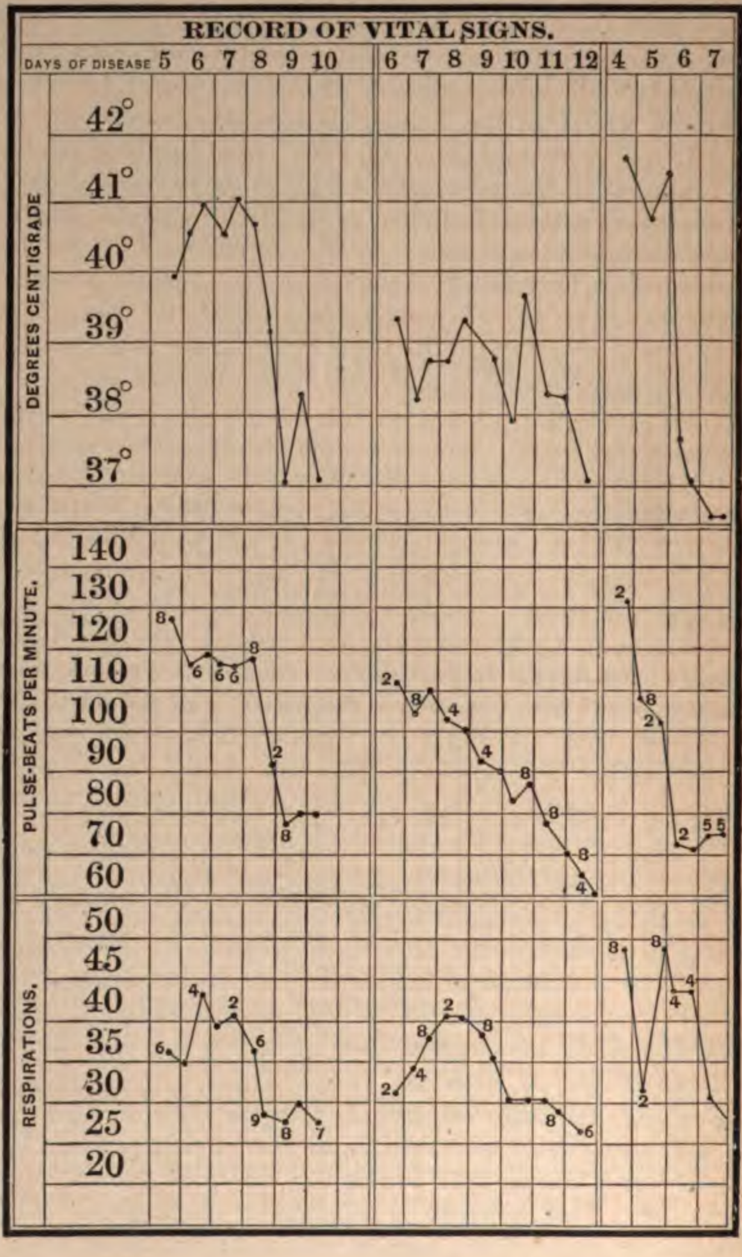
Jan. 29. Subcrepitant râle (râle crepitant redux) is heard over upper lobe; expectoration muco-purulent, hardly at all colored; no fever. Stop mixture.

February. Patient convalesced very rapidly, and only remains in the hospital for the treatment of a malarial element.

In a few words these three cases may be summed up.

But first, the word "defervescence" used in these histories and in the abstract below needs definition. It is new to American medical literature, having first been used by the learned professors of the German schools, to express in one word the cessation or subsidence of the febrile phenomena of disease. It has been very recently adopted in England, and finds a prominent place, as well as the entire subject of thermometry, in Aitken's "Science and Art of Medicine."

In reading the cases and looking over the table of vital signs, the first thing to be remarked is, at what a late period of the disease the patients were received; the most recent on the fourth day. This, however, is a difficulty which attends all investigations made upon hospital subjects; but, fortunately, in other places the disease has been studied so early as to deter-


RECORD OF VITAL SIGNS.
DAYS OF DISEASE
5 6 7 8 9 10
6 7 8 9 10 11 12
4 5 6 7
DEGREES CENTIGRADE
42°
41°
40°
39°
38°
37°
PULSE-BEATS PER MINUTE.
140
130
120
110
100
90
80
70
60
RESPIRATIONS.
50
45
40
35
30
25
20

mine very conclusively that the increase of fever is rapid, and that a very high temperature may be expected within the first twelve hours. In cases I. and III., which were typical of simple acute inflammation of one lobe, the temperature, high at the beginning of the observation, continued to rise, or did not fall except in so far as the regular morning remissions were concerned, until a certain point of the disease had been reached, the ninth day in one case and the fifth in the other, and not till the ascending pathological changes had been wrought; in other words, not until hepatization of the implicated lobe was fully established. This fall in heat, pulse and respiratory movements (defervescence) was complete in case III., the thermometer never again rising above 37° C. In case I., the same was true, with the exception of a single moderate elevation in the evening of the tenth day. Case II. was remarkable for the low intensity of the fever. The defervescence was partially effected on the ninth and tenth days, but during the evening of the latter an alarming increase of 1.75° C. led us to suspect that the disease had invaded a second lobe, when it will be noticed that the pulse and respirations (80 and 30 respectively) gave no warning. Physical examination showed that the thermometer was right. The next morning defervescence occurred fully and finally in the course of two days. It may also be seen how accurately the estimation of the heat determines the cessation of ascending pathological changes, and marks the beginning of those processes by which the vital actions restore the parts affected, and the system generally, to a state of health. That the third period of pneumonia is a resolution, is confirmed by the fact that, in normal cases, the temperature never rises above 37.25° C. after hepatization is complete. Were it true that this disease ends by a suppurative stage, the thermometer would doubtless remain high until the process was completed.

In these two points the diagnosis of disease and of complications, and reliability as an element of prognosis, lies the great value of this means of observation. To go into all the facts necessary to sustain these positions would require more space than can be allowed to a hospital report intended simply to call attention to, and invite trial of, the matter in question. Suffice it to say, that Prof. Wunderlich, of Leipzig, has, with others, so thoroughly investigated the subject before giving it to the profession, that he made no less than half a million careful observations, and ascertained the temperature-variations of nearly all diseases so accurately that his pupils can, by merely looking at the diagrammatic record of a case, almost always correctly diagnosticate the disease without having seen the patient or heard of any other symptoms. He and many other leading physicians make constant use of the thermometer in private practice. The limited trial made in this hospital has not at all lowered the expectations raised by reading the published accounts; in many cases a diagnosis has been arrived at, and a complication detected long before the other objective signs would have enabled us so to do. More especially are the distinctions of fevers into the great types of typhus, typhoid, remittent and intermittent, clearly indicated and not to be mistaken. A last point urged is, that the surface heat, as measured in the axilla, is not liable to variations from the nervous, emotional causes which render the pulse and respiration so very changeable and unreliable. Being the direct result of the molecular changes produced by pyrexia (although the precise relation of the degree of heat to the amount and proportions of the substances resulting from retrograde metamorphosis is not yet ascertained), it cannot be immediately affected by causes acting through the senses, which so disturb other objective signs, for instance, the sudden arrival of the physician, of a friend, of news, the movements frequently necessary to the comfort of the patient, or to facilitate examination, etc.

The whole matter of the utility of medical thermometry is founded upon the fact, that the normal temperature of the human body is invariably fixed within certain limits. Very numerous observations by competent observers have determined this. The following are those of Prof. Traube, of Berlin, the average of many studies of healthy adults at fixed periods of the day, taken in axilla:

36.8° C., 98.24° F.	at 7 A.M.	
37.0° C., 98.69° F.	at 10 A.M.	After breakfast.
37.0° C., 98.65° F.	at 1 P.M.	
37.1° C., 98.78° F.	at 5 P.M.	After dinner.
36.8° C., 98.24° F.	at 7 P.M.	

As to daily practice with the thermometer, the instrument should be an accurately made one, perfect in every respect. Those used abroad (not at present to be had in this country) are graduated to fifths and tenths of degrees, and should be preferred. However, one graduated to degrees only will suffice, if greater care be exercised in reading off the mercury, and a practiced eye may even estimate one-eighth of a degree Centigrade on such a scale. In all cases the thermometer should have an outer glass casing, to protect it from injury and external influences.

The bulb is to be inserted in the axilla, just beneath the fold of the pectoralis major muscle, not too deeply, the forearm of that side carried across the chest, and the elbow secured by an assistant, or by the patient's other hand. It is left *in situ*, carefully isolated from all clothing, and in perfect contact with the skin, for eight, or even ten minutes, being looked at three or four times, the last two determining whether the column of mercury has ceased to rise; the degree (and fraction) is then read off and registered. While waiting, the physician has time to count and record the pulse and respirations, and even to proceed with many other points of investigation. If time be precious, the bulb may previously be heated about to the expected heat and then inserted, when three or five minutes will be enough for a correct estimate. With the exception of the anus, the axilla is found to be the most reliable locality for the purpose.

ABSTRACT OF PROF. L. THOMAS' PAPER.

Early observations:

39.2° C., 102.5° F.	observed	4	hours after	first symptoms.
40.5° C., 104.9° F.	"	9	"	"
40.2° C., 104.4° F.	"	12	"	"
40.6° C., 105.1° F.	"	23	"	"
41.0° C., 105.8° F.	"	36	"	"
41.4° C., 106.5° F.	"	24	"	"

Variations of temperature in regular cases:

Minimum in morning, maximum in afternoon, and again after midnight a decrease.

Types of fever:

1st. In no case purely continued.

2d. Some cases with small differences between the morning and evening measurements (0.25° C. to 0.28° C.) have very seldom been observed, and only for a single day.

3d. In the vast majority of cases the differences amounted to 0.5° C. to 1.2° C.

4th. More than once differences of 1.4° C. have been noted.

5th. A few cases in which almost complete remissions occurred.

The *highest point of fever* may be known by an extraordinary elevation, or by a great fall in the temperature, compared with preceding observations.

The *termination of the fever* (defervescence) generally occurs within forty-eight hours, the temperature sinking to the normal standard. The *days of defervescence* have been carefully noted.

46 CASES BY THOMAS.

Days.	Cases.
2d	2
3d	6
4th	6
5th	11
6th	5
7th	10
8th	4
9th	0
10th	2

107 CASES BY ZIEMSSEN.

Days.	Cases.
2d	0
3d	9
4th	3
5th	31
6th	5
7th	35
8th	4
9th	9
11th	8
13th	3

Thus out of a total of 153 cases, 45 turned on the seventh day and 42 on the fifth. Ziemssen states that in cases in which more than one lobe is involved, the defervescence is likely to be put off until the eleventh or thirteenth day. Thomas is, however, inclinded to doubt this, for in 18 of his cases, in which more than one lobe was affected, he observed but two in which the pyrexia did not subside by the seventh day; and those on the tenth.

Defervescence is affected by the lobes as follows:

Right upper lobe—1 on second day; 4 on seventh; 1 on eighth.
Right lower lobe—2 on fourth; 4 on fifth; 2 on sixth; 1 each on seventh, eighth and tenth.
Left lower lobe—1 on second; 5 on third; 2 on fourth; 6 on fifth; 1 on eighth.

Temperature towards fatal termination:

Majority of cases ending in delirium:

2 on sixth day, 41.4° C., 106.5° F. and 40.9° C., 105.6° F.
1 on seventh, 43.0° C., 109.4° F.
2 on eighth, 41.3° C., 106.4° F. and 40.5° C., 104.9° F.
1 on fourteenth, 42.7° C., 108.9° F.

In one case ending with furious delirium, hallucinations, trismus and tetanus, an elevation of 3.8° C. occurred in six hours. In three cases dying of suffocation (asthenia and apnœa), there was no elevation towards the agony; in two there was great emphysema, and the temperature remained between 38.5° C. and 39.75° C.; in the third, accompanied by a fever of great regular remissions, the last estimate was 37.5° C.

For further details and general information the reader is referred to the *Archiv der Heilkunde*, of Leipzig; to Aitken's *Science and Art of Medicine*, and to articles in the London *Medical Times and Gazette*, for 1858 and 1861.

ON THE SUBCUTANEOUS USE OF SULPHATE OF QUININE IN CASES OF MALARIAL NEURALGIA.*

The following cases are taken from the Medical Case Book of the New York Hospital, to illustrate the effect of quinine injected hypodermically over the seat of pain in neuralgias due to blood poisoning by malaria. Previous to December, 1866, all such cases had been treated by means of quinine and iron, given internally, and blisters put over the affected part. The success of this, the ordinary plan of treatment, as may be seen in cases I., II. and VI., was hardly satisfactory. From three weeks to three months seems to have been the usual time of hospital residence.

The first case related had already been under treatment for five weeks, without benefit, when Dr. Wm. H. Draper, the attending physician on duty, directed the injection of quinia in the manner described. The success obtained exceeded our most sanguine expectations; in four days the pain was relieved, and in four more a complete and permanent cure was effected. Thirteen injections, of 1.15 cc. each, had been employed; some swelling, induration and tenderness remained where the medicine had been introduced, but these soon disappeared.

The solution employed was the one that for more than two months we had been using hypodermically in cases of malarial fever, and was made according to the formula on page 15.

The injections were given in the following manner: A fold of skin being taken up and firmly held between the thumb and forefinger of the left hand, the point of the syringe was introduced at the top of the fold, where a partial anæsthesia had been induced by the pressure; the needle being put in its full length, the solution was forced in gradually as the needle was withdrawn, so as to throw the injection as much as possible into the track of the wound, and to cause the least rupture of the surrounding connective tissue. After taking out the needle, a little circular friction was generally used, with the view of assisting the absorption. The slight hæmorrhage sometimes following the operation was easily controlled by finger pressure.

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* From the *New York Medical Journal*, August, 1867.

The immediate effect of such an injection is a pretty severe burning pain being felt in the part, due, most probably, to the acid and irritating property of the solution. This pain usually passes off within twenty minutes.

Within two hours of the depositing of the injection under the skin, some swelling and induration of the part begin to show themselves, without heat or redness, and apparently owing to the occurrence of a fibrinous exudation. Usually, in the course of a day, this induration reaches its maximum, and afterwards decreases slowly, and disappears within the fortnight. Sometimes, in delicate tissues or when the injection has been given hastily, some degree of ecchymosis makes its appearance in the course of two days. Later, the therapeutic effect of the medication shows itself in the arrest of the pain and hyperæsthesia, followed in some instances by slight though distinct anæsthesia. No abscess or other unpleasant consequence has followed any of the seventy-eight injections used in these six cases.

The *modus operandi* of the hypodermic injections of quinia may be said to be threefold. 1st. By absorption of the drug into the general circulation, whereby it obtains its specific effect, tending to the removal of the cause of the neuralgia. 2d. By the direct therapeutical action of the quinia on the nerves and cellular elements of the part affected. 3d. By pressure; a purely mechanical effect of considerable importance, due to the large quantity of fluid used and to the amount of exudation thrown out.

Case I.—A., æt. 30. A seaman.

Admitted November 5, 1866. Has had intermittent fever (contracted in Aspinwall) more or less for four weeks; last chill being on the 6th inst. Has suffered from pain over spleen; evidently a neuralgia affecting the lower intercostal nerves. The spleen itself is a little enlarged, and tender under pressure. Patient is considerably cachectic. Quinia and chalybeates ordered. Locally, dry cups, tinc. iodine, and blister applied. Dec. 13. Has had no chills, but neuralgia is about as severe as at time of admission. Ordered a pill of quiniæ sulphat., .12; and bellad. extract., .015; to be taken thrice daily.

Dec. 19. Pain is no less; sharp and nearly constant in left hypochondriac region. Stop pills; continue quiniæ sulph., .12 ter in die, and the iron. Ordered local injections of 1.15 cc. solution of quiniæ, night and morning. Dec. 23. No pain to-day. Continued one injection a day. Dec. 28. Stop injections, as pain has not returned. Continue internal treatment. Jan. 7. Patient is discharged cured.

CASE II.—M., æt. 29. A seaman.

Admitted Nov. 2, 1866. During the last two months patient had quotidian intermittent fever, contracted in Aspinwall.

From the first had more or less pain over the region of the spleen, running towards the epigastrium; of late the pain has been quite severe. Patient is cachectic and weak. Ordered quinia in sufficient doses to arrest chills; the citrate of iron, together with good food, and 250. cc. sherry wine. Nov. 19. There is much general improvement, but pain in left side is about the same. Spleen is badly enlarged. Ordered a blister locally. Stop quinia, but continue iron. Dec. 12. Blister has been repeated, but pain remains the same; ordered an injection of quinia, 1.15 cc., injected over seat of pain twice a day. Dec. 15. Neuralgia is gone from the side, but patient is seized with a severe hemicrania of left side. Continued injections and the iron; ordered quinia, .12, ter in die. Dec. 20. Injections were now given in shoulder, near neck. Dec. 28. Hemicrania well; no return of pleurodynia, and no chills. Stop injections. March 2, 1867. Patient has remained, on account of sore throat and conjunctivitis. Is to-day discharged, cured.

CASE III.—C., æt. 31. A seaman.

Admitted February 1st, 1867. Patient eight months ago had a severe attack of intermittent fever. He contracted a diarrhœa at Aspinwall, one month ago. Soon after, he began to complain of a severe paroxysmal pain in the epigastrium.

On admission, the diarrhœa has ceased, but the tenderness and pain continue in the skin over the upper part of the abdomen. There is also a decided tenderness developed by pressure on the sixth and seventh dorsal vertebræ. Feb. 3d. Ordered 2.2 cc. of solution of quinia injected in epigastrium. As patient is decidedly cachectic, he takes bark and iron, with quinia, .12, ter in die. Feb. 8th. Yesterday, neuralgia had ceased; the injection is reduced to 1.15 cc. Feb. 11th. Has no pain; stop injections, but continue internal remedies. March 2d. Is now well. Some induration still remains where injections were put in. Discharged, cured.

CASE IV.—Same patient. Re-admitted April 12th.

He went back to Aspinwall, and, three weeks ago, the pleurodynia returned, locating in the epigastrium, and entire right side of chest, up to clavicle. Patient is quite cachectic, but spleen is only a trifle large, and the liver normal in size. The pain is rather worse at night, but there is no febrile movement. Ordered calomel, .60, followed by 30. cc. ol. ricini. April 14th. Ordered quinia, .12, ter in die, and mixture of bark and iron. Also a hypodermic injection of quinia, .25 (2.2 cc.), every morning, over seat of pain. April 16th. Pain has ceased; continued treatment. May 4th. Patient has had no return of pain. Subsequent to the arrest of the neuralgia he had an attack of dysentery and one of gout; both easily mastered. Is to-day discharged, cured.

CASE V.—S., æt. 32. A seaman.

Admitted April 10th, 1867. Patient comes from a southern port. Has had no attack of malarial fever. For last three months has suffered from severe pain in the right side of back, running up to the shoulder. The liver is slightly enlarged; there is no pleurisy. General condition good. Ordered house mixture of bark and iron, and an injection of 2.2 cc. solution of quinia, each morning, about angle of scapula. April 30th. Hypodermics stopped. Patient has none of his pain. A stitch is occasionally felt in various parts of his chest. May 3d. Is discharged, cured.

CASE VI.—S., æt. 46. A seaman.

Admitted April 23d, 1867. About five weeks ago, patient had quotidian intermittent, which was contracted in a southern port. He had only a few paroxysms, when there appeared a sciatica of the left thigh, which has continued till now. Patient states that he never has had any rheumatism; but, that, one year ago, he had a severe attack of Aspinwall fever.

[In August, 1865, this same man was received into the house, with a similar sciatica, contracted in the same way, which resisted the ordinary anti-rheumatic treatment for three months (by means of colchicum, iodide of potassium and blisters), and finally yielded to quinia and iron, given internally.]

Patient is ordered the mist. ferri et cinchonæ of the house, and hypodermic injections of quinia, .25, in morning, over course of left sciatic nerve, beginning above. May 1st. Almost no pain in thigh. Patient complains of anæsthesia along course of nerve below injection; some pain remains below knee; continue treatment. May 4th. No more injections since yesterday; neuralgia is substantially cured; continue tonics. May 7th. Complains of some pain in right sciatic nerve. May 13th. Had two injections of 2.2 cc. over right nerve, and pain ceased. To-day sent out cured.

In looking over these cases, some instructive points are seen. No doubt can be reasonably entertained as to their nature, without calling into question the existence of the class of malarial neuralgias. These men were all seamen of the southern trade, some going as far as Aspinwall; they all suffered from remittent or intermittent fevers before the onset of the pain; in every one the malarial cachexia was present; they were all, with one exception (Case IV.), free from the rheumatic taint; in all but Case VI. the disease affected the branches of the intercostal nerves, the lower ones by preference; and in these cases careful examination failed to reveal pleuritic effusion or friction sound. In all instances there was a marked hyperæsthesia of the integuments of the seat of the pain; in none anæsthesia. In only two cases (III. and IV.) was tenderness found over the spinal processes, corresponding to the origin of the involved nerves (*apophysal point* of Trousseau). In Case II. a complete metastasis occurred from the left hypochondriac region to the left side of the head and face; and in Case VI. from the left to the right sciatic nerve. In none of these cases was the pain intermittent, nor was it ever of the same intensity during the twenty-four hours, being usually more distinct in the afternoon.

As to the merits of the plan of treatment, to say that it is specific would be a reckless assertion. To deny it very great efficacy would be to reject the testimony of the above cases. That the internal administration of quinia and iron must be kept up during the treatment by injections, and continued for some time after the pain has increased, is evident from a consideration of the pathology of the disease; and that the above-named remedies alone will cure this form of neuralgia is not denied; but the history of the first attack of Case VI., in 1865, and the account of the beginning of cases I. and II., show how often blisters may be applied, and how long tonics may be given without alleviation of the symptoms.

ON TREATMENT OF MALARIAL FEVERS BY THE SUBCUTANEOUS USE OF THE SULPHATE OF QUININE.*

QUINIA seems first to have been subcutaneously employed by Dr. Chasseaud, of Smyrna, who in 1862 reported one hundred and fifty cases of malarial fever thus successfully treated. He claimed greater rapidity of action and economy in the use of a costly medicament as the advantages of the new plan. About the same time a similar attempt was made in France by Goudas,† who published fifteen cases, and Moore ‡ used Chasseaud's method in India. During the winter of 1864–5, Dr. Maury § treated some twenty-five cases of intermittent fever in the rebel hospital at Greenville, Alabama. In 1865 Mr. Craith,‖ of Smyrna, continued to give quinia hypodermically with flattering and constant success, while Desvigne in France, Eulenberg ¶ and Lorent** in Germany, were making some few successful experiments.

This method was first tried in the New York Hospital by Dr. G. M. Smith, the attending physician then on duty, in a case of congestive remittent fever, during September, 1866. It has since been applied to all varieties of malarial fever, and may now be said to be a part of the regular practice of the house.

The solution of sulphate of quinia used in the hospital is made according to the following formula:

Take of subsulphate of quinia, 4.; dilute sulphuric acid, 2.4 cc.; distilled water, 31. cc. Mix. Make a solution and filter with the greatest care.

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*From the *N. Y. Medical Journal*, December, 1867. The material for this and the preceding essay was collected by the author while House Physician of the New York Hospital, 1866–7. [R. W. A.]

† *L'Union Médicale*, 1862.

‡ *Lancet*, August 1, 1863.

§ *American Journal of Medical Sciences*, Oct., 1866, p. 371.

‖ Letter to Med. Chir. Society of London, December, 1865.

¶ Die Hypodermatische Injection der Arzneimittel, etc. Berlin, 1865.

** Die Hypodermatischen Injectionen nach clinischen Erfahrungen. Leipzig, 1865.

2.2 cc. of this are equal to .25 of quinia.

Or the solution may be varied by the addition of .25 or .36 of sulphate of morphia, which combination renders the injection less painful.

The mode of giving an injection of this solution by means of an ordinary hypodermic syringe, has been fully detailed on page 10.*

At first less acid was used, for fear of causing irritation, but it was soon discovered that the real causes of abscess were the undissolved crystals of quinia and the particles of dust which imperfect closing of the bottle allowed to drop into the fluid. It was also found that an excess of acid removed the most fertile source of danger, while it but slightly increased the pain of administration. In reality, there is but little more acid in the above formula than in Moore's, for he used twelve drops of the *pure* acid to dissolve 2. of quinia in 16. cc. of water. Dr. Maury, of Alabama, employed a solution, 4. cc. of which contained .50 of the salt.

From the fact of sailors being received into this institution as patients, malarial fevers form a large part of the practice of the house. These cases come in mostly from the various ports of our own Atlantic and Gulf coasts, and from Cuba, the West Indies, Mexico, and Central America. In many of these ports (Aspinwall, Mobile, and Wilmington being the worst) the poison is most intense, and produces not only severe attacks of fever proper, but also deep and long-continued cachexia. Congestive cases come in during the summer and autumn from the three above-named places, and are of a very fatal character. These peculiarities of the fever necessitate the exhibition of much larger doses of the anti-periodic medicine than are usual in Northern practice. Besides the amount of quinia required for the breaking of the attack, the prolonged use of small doses combined with iron is resorted to, in order to correct the cachexia.

The usual treatment of these cases has been as follows for the past few years:

A simple intermittent of the tertian type was cut short by the giving of three doses of .30 of quinia two hours apart, and so calculated that the same interval of time should intervene between the last dose and the expected chill.

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* *New York Medical Journal*, August, 1867, p. 403.

A quotidian usually required four such doses given in a similar manner. In both forms, subsequently to the stoppage of chills, some .36 of quinia were given daily, together with some form of iron;[*] on the seventh, fourteenth, and twenty-first days (counting from date of last paroxysm) .30 extra were exhibited about two or three hours previous to the chill time.

Simple remittent fever was treated by means of .12 doses of quinia given every two hours until the headache became very severe or the fever ceased. In many cases the medicine could be continued for twenty-four or forty-eight hours, when the force of the fever being broken, the doses were gradually reduced until, in convalescence, .12 three times a day, combined with a chalybeate, were ordered, and continued up to complete recovery. Severe cases, simple, or tending to the congestive form, were treated by hourly doses of .12 or .30 pushed until cinchonism became apparent. Of course, it is understood that this account only relates to the quinia, its mode of administration, and quantity employed, making no mention of the numerous other means resorted to, such as purgatives, local applications, stimulants, and food.

When it was decided to use the hypodermic method it became necessary to work out rules for giving the remedy, both as to time and quantity, no explicit directions being found in any of the published accounts at hand. One single statement there was to serve as guide, that .06 of quinia under the skin was equivalent to .30 or .36 by the mouth, (Moore). In the course of a few experiments it was discovered that this was far too high an estimate, at any rate for the class of cases coming from the South. It was observed that .25 of the medicine were needed to break an attack of tertian fever, and that fully .50 were required for a quotidian. In congestive cases, of course, the amount injected varied according to the severity of the attack, and it was always given upon the estimate that .06 subcutaneously equaled .25 by the mouth. By following the experience gained while giving mouth-doses, the injections were at the first given two hours before the time of the expected paroxysm, and this was found so successful that the rule is now invariably followed. Where two injections were required, if there was time to spare, .25 were given four and the other dose two hours before the expected chill. If the time was too short, both injections were given at once.

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[*] The "black mixture," so much used in the hospital as a tonic, is made according to the following formula: Ferri ammon. citrat., 1.; tinct. cinchon. co., 31. cc.; aquæ, 95. cc.. M. 15. cc. at a dose.

The following histories will illustrate the working of the method in intermittent fever:

Case I.—B——, a seaman, æt. 28, admitted June 22, 1867. Comes from Florida and has had quotidian chills for eight days; paroxysms at noon, retarding a little. On admission is rather feeble. June 23.—To-day had a hypodermic injection at 10 A.M. (.25), but at 1 P.M. had a severe chill. 24th.—No chill to-day. This morning had .25 at 10 A.M., and the same at 11 A.M. Ordered mist. ferri et cinchon. of house, 15. cc. thrice daily. July 1.—Yesterday had .25 under the skin at 11 A.M. No chill. Continue mist. ferri. 3d.—Is sent out well.

Case II.—V——, a seaman, æt. 19, received July 17. Comes from Savannah and has had an attack of tertian ague twelve days. Last chill occurred at 4 o'clock P.M., yesterday. General condition is fair; bowels are open. July 19.—Yesterday had .25 of quinia under skin at 1 P.M. (as chills anticipate a little), and had no paroxysm. Ordered mist. ferri et cinchon., 15. cc. thrice daily, and as there is some cachexia, quin. sulph. .12 thrice daily. 24th.—Yesterday being weekly anniversary of chill, had .25 subcutaneously at 1 P.M. Is much improved in appearance and has had no chills. Discharged cured.

Case III.—W——, a seaman, æt. 22, admitted April 23, 1867. Patient has had a quartan intermittent fever contracted in East Indies, fifteen months, off and on. Has never been free from chills for more than six weeks at one time. He states that from the first every chill has been accompanied by a crop of nettle-rash. On admission, general condition is pretty good, though the spleen is a little enlarged. On 21st had a severe chill at 3 P.M. April 25.—Yesterday, at 3.30 P.M., had a severe chill. About twenty minutes before attack he called attention to an eruption, clearly of urticaria, covering the entire body, and causing intense itching and burning. His pulse was then accelerated and the axilla heat was about 39.5° C. To-day takes quin. sulphatis, .12, thrice daily, and the tonic iron and cinchon. mixture. 28th.—Yesterday had quinia, .25, hypodermically at 11 A.M. and 1 P.M., but chill occurred about 5 o'clock, accompanied, as usual, by nettle-rash. Continue small mouth-doses and mist. in interval. May 1.—Had injections of .25 at 10 A.M., 12 M., and 2 P.M. yesterday, and escaped chill. Continue tonic treatment. May 26.—Patient has had no return of chills. Had quinia, .25, on anniversary days (May 4, 11, 18 and 25,) and has kept up black mixture. Region of spleen has been occasionally painted with tr. iodine. Since 14th inst. has also taken a little of Fowler's solution. 27th.—Is discharged at request, nearly well.

These three cases give a correct idea of the general management of the common intermittents. In many instances no quinia was given after the last chill, excepting the .25 on anniversary days, thus making an immense saving in the use of the medicine, not more being employed in the entire course of many cases than was formerly needed to avoid a single paroxysm. Up to August 6, thirty-five such cases have been treated with perfect success.

In the remittent form, the experience is small and not satisfactory. In two cases, from one to four injections were given in the remissions, but with little effect on the fever, while the number of injections caused considerable annoyance to the patients. Although the experiment has not been repeated in the hospital, yet further trials should certainly be made, when a time and mode of giving the medicine may be found which shall lead to success.

In about ten congestive cases the results have been satisfactory, though, as usual, the mortality has been very great. In only one or two cases have the injections been used alone, being usually employed as an accessory to the ordinary treatment of quinia and whiskey given by the mouth. The following case, reported at the time by Dr. H. D. Bulkley* (the treatment of which was personally attended to by the writer), is an interesting one, and is worth reproducing here as illustrative of the new method:

"——, a stout, well-developed sailor, forty-four years of age, was brought into the hospital at 8 A.M., October 8, in a state of collapse from a chill the night before, from which he had not rallied. His skin was cold and clammy; pulse could not be felt at the wrist; considerable dyspnœa and anxiety, with some laryngeal hoarseness; tongue slightly coated, white, pale, and quite cold. He was conscious and rational, but could only speak in a whisper, and there was great thirst. As the stomach was not irritable, 15. cc. of whiskey was given every half hour, and ice to allay the thirst. The treatment was commenced by a stimulating enema, dry cups to chest, and sinapisms to the nape of the neck and legs; and to avoid irritating the stomach, and for speedier effect, quinine in solution was injected under the integument of the upper part of the chest—.25 at 8.20, 9.20, and 11 A.M., and 1, 2, and 3 P.M.; and about the middle of the day three doses of .30 each were given by the mouth at intervals of half an hour; but as this disturbed the stomach it was discontinued. In the afternoon dry cups were again applied, the use of stimulants being continued. At 4.30 and 6 P.M. the hypodermic injections were again given. At 8 P.M. quinine was given by the mouth, .30 every hour. The amount of quinine given hypodermically from 8.20 A.M. to 6 P.M. was therefore in all 2.15, estimated to be equivalent in therapeutic value to a little over 8. by the mouth.

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* *New York Medical Record.* Vol. 1, p. 489.

"At 3 the next morning (October 9), at the end of nineteen hours, his pulse was perceptible, and he became comfortably warm. His voice was now stronger, but it, as well as the breathing, indicated larnygeal trouble. Nothing abnormal, however, could be seen in the throat, nor could anything be felt by the finger. The quinine was continued every hour, and the whiskey reduced to 15. cc. every hour, and beef-tea ordered freely during the day. At 8 P.M. he had fully rallied, though there was considerable dyspnœa and restlessness, and quinine was reduced to .30 every two hours. In thirty-six hours the patient had taken 2.15 hypodermically and 12. by mouth of quinine, estimated to be equal in therapeutic value to 15.5 of the salt. He progressed favorably until the 14th (five days), reaction having been fully established ; and both the quinine and whiskey having been reduced in quantity. Ecchymosis had taken place at one or two points where the injections had been made. On that day, however, he became worse. Obscure symptoms occurred, made up of laryngeal obstruction and pulmonary and cerebral congestion. Stimulants and quinine were now stopped, and 60. to 90. cc. of blood taken from the nape of the neck. At midnight he became quite restless, and persisted in rising to have a stool near the bed, refusing to use the bed-pan, when he almost instantly sank back and died, apparently from syncope. No autopsy could be obtained."

This patient was undoubtedly saved from death during the algid period by the quinia, and he only died from the effects of the secondary disorganization of the blood so common after severe attacks of the fever. It is noteworthy that he never had a symptom of cinchonism.

In conclusion, a word as to certain consequences likely to result locally from the injections. Abscesses and phlegmonoid swellings have been observed in the hospital. In all, out of a very large number of injections (one patient receiving over one hundred), only six abscesses occurred, and not one of these after the middle of last January. In each instance the result was attributable to the introduction of insoluble particles along with the fluid. Maury reports two abscesses out of twenty-five cases, and Moore the same number out of thirty. When trouble was about to ensue, the swelling normally consequent upon the injection increased after the second day, and redness, together with a beating pain, were noted. The centre of the swelling was usually soft from the first, and at that place the pointing of the pus occurred. These phlegmons were first poulticed and then opened early by a free incision. No difficulty was observed in their healing.

Since January there have occurred a small number (not more than six) of what have been called phlegmonoid swellings. In these, the swellings, redness and heat were present, but the beating pain was not, the patients complaining only of a sensation of burning. As early as the soft centre became distinct, an exploring-needle was introduced, letting out a small quantity of serum; after which the swelling was again poulticed. In all these cases complete resolution occurred.

In numerous instances the patients complained of temporary anæsthesia in or about the part where the injections were given; but the place of election in fever cases being the chest, abdomen, and back, no great inconvenience ensued. Ulceration, sloughing, and reflex nervous disturbance were never observed, nor was cinchonism ever clearly produced.

THE HYPODERMIC INJECTION OF QUININE.*

TO THE EDITOR OF THE MEDICAL RECORD.

Sir: In your issue of August 1st, 1870, there appeared a letter from Dr. Stephen Rogers, in which that gentleman dwells upon the danger attending the hypodermic use of the sulphate of quinia. The doctor speaks of the physicians of the New York Hospital as having expressed opinions upon the subject. I must confess that I have failed to find in the medical journals any communication touching this matter, by any of the six gentlemen of the medical staff of that institution. However, on reading Dr. Rogers's paper in the "Transactions of the American Medical Association, for 1869," I find that he refers to reports published by me in the *New York Medical Journal.*† It may be proper to state that these reports were in no wise *inspired* by any of the attending physicians of the New York Hospital, and that no one but myself is responsible for any opinion or statement therein put forth. There are many points in Dr. Rogers's paper which might call for criticism; but my object is simply to state certain facts and opinions expressed by men experienced in the almost daily use of hypodermic injections of quinia, bearing upon the main point at issue, viz.: the supposed danger of acid solutions used hypodermically. This is, it strikes me, a matter of importance in the whole field of hypodermic medication, and it is important to know if there be any evidence proving that a moderately acid solution, thrown under the skin, causes abscess, tetanus, or other grave disturbances. I may here state that not one word in the doctor's letter, or in his paper in the "Transactions of the American Medical Association," 1869, indicates that he has ever given a single hypodermic injection of quinia! How far this circumstance qualifies Dr. Rogers to sit in judgment upon the question at issue, I leave to the readers of the *Record.*

A case of tetanus occurring after a hypodermic injection of quinia, related by Dr. Sale in the *New Orleans Medical Journal*, is referred to by Dr. Rogers, and the *post hoc ergo propter hoc* indorsed. We are not afforded proof that no nerve was wounded in administering the injection; that no solid particles (crystals of quinia or foreign bodies) entered the subcutaneous connective tissue; nor that the climatic conditions were unfavorable to the development of the neurosis—all points, in my opinion, worthy of investigation before deciding that the tetanus was "produced by the administration of quinia hypodermically."

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* From the New York *Medical Record* of Oct. 15th, 1870.

† On the Subcutaneous Use of Sulphate of Quinia in Cases of Malarial Neuralgia.—*New York Medical Journal,* August, 1867.

On the Treatment of Malarial Fevers by the Subcutaneous use of the Sulphate of Quinia.—*New York Medical Journal,* Dec., 1867.

The report of the committee of the Royal Medical and Chirurgical Society * is most unsatisfactory, and fails to meet the requirements of modern experimental physiology. The committee do advise against the use of acid or strongly alkaline solutions; while nothing in their report appears to show that a well-made acid solution produces inflammation. The abscess which they had to treat, after an injection of quinia, was provoked by the introduction of 30 cc. (!) of solution into the areolar tissue. Nowhere do they refer to the necessity of carefully filtering solutions, or to peculiar care in the cleansing of the syringe. Those who first employed the method—Chasseaud, Moore, Desvigne—were unfortunately obliged to use very imperfect syringes, or even to introduce the solution of quinia through an incision; consequently, the local troubles which they had to deal with cannot rightfully be brought into this inquiry.

Now for the opinion of more recent writers on the subject. Chas. Hunter (*Lancet*, 1863, vol. ii., p. 444), speaking of hypodermic injections of quinia, says: "But is there no fear of abscess or diffuse inflammation following the puncture? So frequently have I asked the question, that I may take the present opportunity to reply: No, certainly not." He then speaks of various precautions to be observed in attempting this method, and adds, *without giving any facts to support his position*: "To employ a fluid otherwise than neutral would also irritate. True, some fluids cannot be made perfectly neutral, but so nearly so that no irritation occurs." The difference between a nearly neutral and a slightly acid fluid is rather difficult to ascertain, I imagine. An even greater authority, probably the best writer and experimenter on hypodermic medication, A. Eulenberg, is even more explicit in his statement.† On page 40 he states that he has never seen local inflammatory trouble follow hypodermic injection of quinia in his own practice; adding, that in the practice of a colleague, a swelling, at first painful, lasting one month, ensued after the injection of a "turbid" solution. And yet Dr. Eulenberg's formula is so like that of the New York Hospital that it must be startling to one who sees in dilute sulphuric acid a fertile cause of abscess, tetanus, etc. Eulenberg directs quiniæ sulph. 4.; acid sulph. dil. q. s.; aquæ distil. 31. cc.; to be mixed and filtered. How far the q. s. in this solution differs from the 2.5 cc. in that used at the New York Hospital, I leave to pharmaceutists to determine.

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* *Medico-Chirurg. Trans.*, 1867.

† Die Hypodermatische Injection der Arzneimittel, u. s. w., Berlin, 1867.

In the New York Hospital, quinia was first used hypodermically in Sept., 1866, and this "abuse" of quinia was continued until the breaking up of the institution in Feb., 1870. At first, all cases treated in this way were recorded with care; but during the last two years of this period hypodermic injections were looked upon so much as a matter of routine, that, although very frequently employed, there are but few cases entered in the case-books of the hospital.

I take the present opportunity of saying, that to Dr. W. Thurman, resident physician in 1868, is due the credit of making the formula, and of laying down many of the important rules for the administration of the solution. The following is the result of my search through the case-books of the four years, giving the *minimum* number of cases recorded in those books, and the number of abscesses occurring. The latter has been the subject matter of oral and written questions addressed to all the residents on duty during the four years, so that the abscess list is, I believe, absolutely correct.

W. Thurman, Res. Phys. April 1 to Dec. 1, 1866; 70 injections, 2 abscesses (both in one case).

E. C. Seguin, Res. Phys. Dec. 2, 1866, to Aug. 7, 1867; 318 injections, 4 abscesses (all in one case, and previous to Jan. 15th); one patient received at least 106 injections, taking quinia as a tonic, in this manner, without having a single abscess or local trouble of any sort. This case alone should decide the question of the danger of acid solutions, as the subject was extremely anæmic and feeble, and was therefore well disposed to inflammatory manifestations. It was during the period of eleven months that the six "phlegmonoid swellings" occurred, and were successfully treated.

T. Skelding, Res. Phys. Aug. 8th, 1867, to April 8th, 1868; 115 injections, 1 abscess (fluid thrown into cutis proper).

J. Vanderpool, Res. Phys. April 9, 1868, to Dec. 1, 1868; 62 injections, 2 abscesses.

[Dr. V. writes me that a very large number of injections were given in this period.]

A. H. Buck, Res. Phys. Dec. 2, 1868, to April 1, 1869; 4 injections, no abscess.

[Dr. B. tells me that during his service hypodermic injections of quinia were very frequently given.]

L. D. Bulkley, Res. Phys. April 2 to Aug. 2, 1869; 2 injections recorded (many given), no abscess.

G. A. Hathway, Res. Phys. Aug. 3, 1869, to Feb., 1870; 22 injections, no abscess.

The total number of injections recorded on the books is 593; the number of abscesses recorded and recalled is 9.

I think, moreover, that the above-named gentlemen would concur with me in estimating that at least one thousand injections of quinia were given at the hospital from September, 1866, to February, 1870. It is noteworthy that *six* of the nine abscesses occurred within the first four months of the practice, and in *two* cases. The above residents, without exception, express it as their deliberate opinion, that the sulphuric acid in the solution is not, *per se*, a cause of local inflammatory trouble. With this view I concur; and go further, with my friend Dr. Thurman, in thinking that the acid is, by insuring perfect solution of the crystals, a source of safety. The ex-residents assign to various causes the production of the abscesses falling under their observation, such as undissolved crystals of quinia, foreign particles in the solution (dust, fungi, sealing-wax), haste in the operation, the throwing of the fluid in the cutis, and want of care in cleaning the syringe.

After offering this evidence to the readers of the *Record*, I feel that something should be added concerning the method of giving the solution employed at the New York Hospital. The formula for the solution was: "Take of sulphate of quinia, 4.; dilute sulphuric acid, 2.5 cc.; distilled water, 31. cc. Mix; make a solution and filter with the greatest care." 2.3 cc. of this solution contain .25 of quinia.

(*a*.) The solution was often inspected, and re-filtered when required. The hypodermic syringe was cleaned with water *always* (at least during my own term of office) before or after use.

(*b.*) Of course, in a hospital receiving a large number of cases of malarial disease, as did the New York Hospital, the syringe is used so much that there is little danger of rust or dust settling on any of its parts. In private practice too much care cannot be taken in cleaning the instrument often.

(*c.*) The following rules for the operation, given in my first report, I believe still to be good: "A fold of skin being taken up between the thumb and forefinger of the left hand, the point of the syringe was introduced at the top of the fold, when a partial anæsthesia had been produced by the pressure; the needle being put in its full length, the solution was forced in gradually, as the needle was withdrawn, so as to throw the liquid as much as possible in the track of the wound, and to cause the least possible rupture of the connective tissue. After taking out the needle, a little circular friction was generally used, with the view of assisting absorption. The slight hæmorrhage sometimes following the operation was easily controlled by finger-pressure."

Judging from experience, I unhesitatingly recommend this method of giving quinia in the following circumstances: 1st. In general hospitals, civil and military, when economy in the use of quinia is a *desideratum*. At the New York Hospital no saving was effected on the whole, in the opinion of Mr. Johnson, the very capable apothecary of the hospital. This was owing to a number of causes, which I cannot enter upon here, but which do not prevent me from saying that the method *may* be made economical. 2d. In private practice, in cases of congestive fever, when quinia cannot be administered by the mouth, or when rapid action is wanted. I do not advocate this to the exclusion of the injection of the remedy into the rectum, as recommended by Dr. Rogers, but would remark that well-directed experiments are needed on this point. 3d. In private and hospital practice, for the treatment of neuralgia of malarial origin, in which affection remarkable results are obtained by local injections of quinia; the drug acting in its specific manner on the system, and locally by counter-irritation. Lastly, I may state that Dr. L. D. Bulkley has informed me that, while resident in the hospital, he several times observed a rapid improvement in the pulse of patients affected with a partial collapse subsequent to severe hæmoptysis, after the simultaneous injection of two, three, and even four full doses of quinia (2. 2 cc.) under the skin.

A STATEMENT OF THE APHASIA QUESTION, TOGETHER WITH A REPORT OF FIFTY CASES.*

IN 1861 the *Société Anatomique* of Paris was startled by the announcement of Prof. Paul Broca, that the faculty of articulate language was to be located in the third frontal convolution of the left hemisphere of the brain. In proof of this proposition he presented two remarkable specimens, obtained at the death of old aphasics, and in the course of the same year other corroborative autopsies were made. But, earlier in the year, the subject of aphasia (with localization in the left anterior lobe) had been the occasion of an interesting debate in the *Société d'Anthropologie*, and at that time very strong negative cases had been cited. In the memorable discussion which took place in the Imperial Academy of Medicine, in 1865, these negative cases were largely increased in number and force. Besides, during the past six years, numerous instances of disease have been published in the periodicals, favoring and opposing Broca's view; so that to-day the question is involved in nearly as much obscurity as ever, and an urgent necessity exists that the profession should turn its attention to the study of cases and pathological specimens, with the view of attempting the settlement of the many vexed points. If the problem can be solved, medicine will not be the only gainer, but a fresh impulse and a new direction will be given to the study of the mind.

The cases which are appended were taken from the case-books of the New York Hospital. Four of them (I., II., III. and IV.) were observed by the writer during his service as assistant and resident physician in that institution. The remaining forty-six cases were recorded before aphasia was recognized, and they are therefore very imperfectly noted, and are open to challenge. But it is to be understood that they are offered only as bearing upon the grosser points of the question. It may be here stated that six probable cases were rejected because of the insufficiency of the record, and two in which speech was much disturbed, on account of a contradiction as to the side on which the palsy existed.

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* From the *Journal of Psychological Medicine*, N. Y., Jan'y, 1868.

Aphasia, alalia, aphemia, are three terms of Greek derivation which in the last forty years have been employed to designate that mental condition, characterized by abolition or abridgment of the function of language, without difficulty in articulation and without a general affection of the intellect.

The word aphasia, although revived by Trousseau, is a very ancient term for mutism. It occurs [Falret] twice in Homer, and is used by a later Greek writer, Sextus Empiricus, who defines it almost exactly in our modern pathological sense. Alalia was employed by the older physicians to designate loss of speech and voice without distinction, until the last century, when Sauvages, Cullen, Swediaur, and the two Franks, separated alalia from aphonia. Bouillaud, in his first communication (1825) adopted the word, but it has since fallen into disuse. Aphemia, a word created by Broca in 1861, was soon rejected on account of its ambiguous derivation; as it might, for instance, signify a bad reputation.

The history of the growth of the aphasia question may be briefly stated.

The impulse was given by the great anatomist Gall,[*] by the announcement in 1809, that there did exist a faculty of language, and that it had its seat, its physical organ, in the anterior lobes of the brain. He attempted to fortify this position by physiological arguments and pathological illustrations. In 1825 Prof. Bouillaud[†] supported this hypothesis by the citation of numerous cases of injury and disease affecting this part of the encephalon, resulting in loss of language. He went further in psychological analysis than Gall, recognizing the elements of memory of words and that of co-ordinated movements necessary to the formation of articulate sounds. In 1836 Dr. Marc Dax, (of Sommières, Dept. of Gard, France), addressed to the medical congress of Montpellier a most remarkable work[‡] embodying his own observations from the beginning of the century, together with many from diverse authors, in which he went far ahead of his predecessors, and claimed that the faculty of speech was localized in the left anterior lobe. It is most extraordinary that this memoir, which entitled its author to the priority in the investigation of the subject, remained absolutely unknown until nearly thirty years later. In 1836 and 1848 Bouillaud again advocated his theory, in communications to the *Académie de Médecine*. A report upon a memoir of Dr. G. Dax (a son of the other Dax), in the *Société d'Anthropologie* of Paris, in the spring of 1861, brought about a most animated debate between Bouillaud and Auburtin in defence of localization; Lelut, Gratiolet, and others against the hypothesis. One of the results of this discussion, was what M. Bouillaud afterward called the "brilliant conversion" of Broca, who brought forward his specimens,[*] and carried localization to its extreme limit; asserting that the organ of speech lay in the posterior part of the third frontal convolution of the left anterior lobe. The clinical lectures of Trousseau at the Hôtel Dieu in 1864 were very valuable contributions to the subject; he being the first to give prominence to the amnesia theory of aphasia. During 1865 the French Academy of Medicine was the scene of an almost violent discussion on the subject, extending over many meetings, at which the anatomical and psychological aspects of the question were debated with great learning and eloquence. The volume of London Hospital Reports for 1865 contained a report of thirty-six cases, by Dr. J. Hughlings Jackson, in which he declined deciding for or against localization. About the same time quite a number of articles on aphasia appeared in European medical periodicals; the two principal being by Moxon in the British and Foreign Medico-Chir. Review,[†] and Auburtin in the Gazette Hebdom. for May, June and July, 1863.[‡] In the British Journal of Mental Science, January, 1867,[§] Dr. Alexander Robertson claimed that the essential lesion was a *motor* and not a *mental one*. In this country a few cases have been reported by Dr. Austin Flint, Sr., in the first number of the New York Medical Record,[‖] and by Dr. Richardson in the Buffalo Medical Journal (quoted in Richmond Medical Journal, May, 1867). On December 21st and 27th, Dr. C. E. Brown-Séquard, in the course of lectures delivered before the New York Academy of Medicine, expressed the opinion that aphasia was a reflex phenomenon. During the month of May, 1867, Dr. H. B. Wilbur, Superintendent of the New York State Asylum for Idiots, read before the Association of Medical Superintendents of American Institutions for the Insane, an interesting paper on aphasia, in which he considered some of the aphasiform symptoms presented by the pupils under his care.

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[*] Recherches sur le système nerveux. Paris, 1809; and Anatomie et Physiologie du système nerveux, etc. Paris, 1810–19.

[†] Traité de l'encéphalite, Paris.

[‡] Lésions de la moitié gauche de l'éncéphale coincidant avec l'oubli des signes de la pensée.

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[*] Sur le Siége de la Faculté de Langage Articulé, Bull. de la Soc. Anat. Paris, 1861.

[†] 1866, Vol. 38, p. 41.

[‡] Tome x., pp. 318–348, 397–455.

[§] Vol. xii., p. 503.

[‖] Vol. i., p. 4.

Aphasia may vary in degree from the forgetting of a few words to the sad extreme of total deprivation of the power of expressing ideas. The former state, consisting, according to Trousseau, solely in the amnesia of words, is well exemplified by Case II., in which at the time of admission not more than a dozen lacunæ could be discovered in the vocabulary. Pliny notes the case of the orator Messala Corvinus, who only forgot his own name. In complete aphasia there coexist amnesia of words, amnesia of written speech, amnesia of gestures. In some intermediate cases the patient can write; in others gesticulate; in anomalous instances the power of ciphering or writing music (Lasègue) has been preserved when ordinary writing was impossible.

In some cases of incomplete and in nearly all complete cases of aphasia, involuntary sentences are ejaculated. A reverend gentleman, affected with an amnesia of words, was forced to add, after the sentence, "Our Father who art in heaven," the words, "let him stay there." Another case in point was that of a lady seen by Trousseau, who being totally aphasic without paralysis, would rise on the coming of a visitor, receive him with a pleased and amiable expression of countenance, show him a chair, at the same time addressing to him the words "*cochon, animal, fichue bête;*" French words hardly allowed by drawing-room usage. Occasionally the ejaculations are meaningless sounds, "cousisi," "menomomenlif," "tau" (the only sound made by Broca's celebrated patient). Women are apt to make use of plaintive or semi-religious expressions, "O dear," "good Lord," etc. Men of the lower classes may retain oaths remarkably. In all such cases, when the attempt is made to teach new words by constant repetition, the almost invariable response is the word or sound retained by the patient, and which may be termed the aphasic echo; the spoken echo.

In cases of medium severity some peculiarities are to be observed in the writing. One or more words are put to paper by the patient, and this written echo often differs from the spoken one. The man who said "cousisi" always wrote "paquet." When right hemiplegia exists, writing should be taught to the left hand in order to investigate the case fully.

To the statement that gesture is totally abolished at times, exception must perhaps be made for the very expressive movements of the eye, which have not been deficient in any case hitherto reported. In case III., while not a sign could be made by the limbs not paralyzed, the patient's intelligence, sorrow, and impatience were well shown in the glance.

The state of the intelligence in aphasia has been variously estimated by different observers. When there is merely amnesia of a few words, the mind seems to be intact in every other particular. In many cases of a complete loss of language, business matters may be attended to, amusements may be enjoyed, games requiring memory of ideas and of facts together with judgment may be played. Later, however, in cases where the brain lesion has been severe, progressive imbecility may develop itself; probably due to softening of the cerebral substance. Dr. Robertson seems to consider as a sign of impairment of the mind, the fact that patients after repeated failures to answer questions correctly manifest grief and weep. It might be suggested that persons with full command of language, and whose mental integrity even Dr. Robertson would hardly dare question, often show equal sorrow at the loss of functions or parts of the body of much less importance than speech. It would perhaps not be exaggerating to say that the opposite state, insensibility to so great a calamity, should be deemed a sign of mental degeneration. Reading, as a test of the integrity of the intellect, may be reasonably objected to, for the reason that there being complete forgetfulness of words, the written or printed page is thereby rendered useless. It is as if reading a page of Hebrew were deemed a measure of the mind of an ordinarily educated American. In a medico-legal point of view this question of intelligence is a most important one, and one which from its very terms can receive no general solution. Each case must be judged of by itself after a careful examination.

The causes of aphasia may be studied under two heads:

1st. The anatomical cause. In the majority of cases, especially such as occur in persons past the prime of the life, apoplexy must be held accountable for the lesion. This effusion of blood may be small, producing only amnesia of speech and writing, or the laceration of brain substance may be of such extent as to cause hemiplegia, and in some cases to be followed by death. In younger persons, if a valvular cardiac lesion be diagnosticated and if the general health be such as to exclude atheroma of the arteries, then plugging (embolism) of one of the branches of the circle of Willis has probably occurred. It is supposed by some that syphilis may lead to a roughening of the internal coat of the arteries, and thus produce coagulation of the blood (thrombosis) which shall deprive a part of the brain of its proper supply of food. Syphilis may, in still another way—the pressure of an internal node upon the brain—develop aphasia. Wounds of the skull and cerebral substance occasionally lead to the same result. Secondary pyæmic abscesses might produce the symptom, though no case thus caused has yet been reported. And, lastly, it must not be forgotten that aphasia without lesion of the cerebral substance has been reported in three instances: by Gairdner,* by Hillairet, † and in Case L of this paper. Whatever may be said of the latter case, that related by the distinguished Glasgow professor must be accepted. It may be added that at the *Société d'Anthropologie* in 1861, M. Rufz stated that he had observed aphasia supervening on the bite of a certain snake *(serpent fer-de-lance)*. This loss of speech was sometimes instantaneous, but usually it came on in a few hours after the accident. In persons who did not die poisoned, the aphasia persisted indefinitely, and it seemed to be entirely independent of the location of the bite. Intelligence was in all cases preserved, and the affected persons went about their ordinary occupations in silence. *Where* all this occurred was not stated. And M. Brown-Séquard has record of more than one case of aphasia due to peripheral irritation, and in which no cerebral lesion could be discovered after death.

2d. Psychologically speaking the difficulty may be of various nature. In order to enter into the necessary details with clearness, let the following be accepted as hypothetical elements of language. To support this division would require greater space than can be allowed to a mere report. Suffice it to say that it is an extension of the views of speech held by Fournié‡ and Wilbur.* The elements, then, that enter into the physiological growth of language are as follows:

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* *Glasgow Med. Journal.* June, 1866.

† *Journal de Médecine Mentale.* Sept. et Oct., 1865.

‡ Phys. de la Voix et de la Parole. Paris, 1866.

a. The sounds or signs, representing or suggesting an idea, transmitted to the sensorium by means of the ear, the eye, or the touch.

b. The formation of the idea (most probably in the gray cortical matter).

c. The memory of the words or signs necessary to express this new-born idea.

d. The willed movements of various parts; organs of speech for phonation, of the hand for writing, and of numerous muscles for gesture.

e. The production and modulation of sound.

a. The sounds or signs. The study of this division may with propriety be omitted, as not coming within the scope of this paper; though in the aphasiform troubles of idiots † and deaf-mutes it plays an important part.

b. The formation of ideas. Is it diminished, and are trains of thought imperfect in aphasics? This important question has received various answers. In examining patients with complete aphasia and hemiplegia, it has appeared to most observers that this part of mental action had considerably deteriorated. But it must be borne in mind that the communication between the physician and the patient is in these cases very imperfect, so that even if there were no testimony rebutting this opinion, it would be well to take it with caution. But there is testimony of the most valuable kind in the shape of accounts given of their own cases, by two distinguished members of the profession. Let the medical witnesses speak. Prof. Lordat, of Montpellier (France), a most attractive extemporaneous speaker, and an excellent teacher, was deprived of articulate and written language for several months in the year 1828. He says: "I thought of the Christian doxology, '*Gloire au Père, au Fils, et au Saint-Esprit*,' and it was impossible for me to remember a single word of it." Again: "I could think upon abstract matters, combine and distinguish them, without having any words to express them, and without paying the least attention to expression. I experienced no embarrassment in thought. For many years accustomed to the trying duties of public teaching, I congratulated myself upon being able mentally to arrange the various propositions of a lecture, and to change the order of ideas at will." Trousseau, not willing to abandon the philosophic doctrine that words are indispensable instruments of thought, expresses the opinion that Lordat must have deceived himself. A professor of the faculty of Paris, confined in bed on account of an injury, was reading the *Entretiens Littéraires*, of Lamartine, when, without other warning, he noticed that he did not fully understand the sense of the text. He dropped the book a moment, then tried again to read, and once more observed that he understood nothing of the light and entertaining pages. Trying to speak, he found that he could not utter a single word: he attempted to write, and failed as completely. Thoroughly alarmed, he proceeded to an examination of his body: moving his tongue and limbs, he came to the conclusion that there was no paralysis. Lastly, he entered into a mental speculation as to what portion of his brain might be damaged. Now, M. Trousseau attempts to show that because he could not understand the *Entretiens*, the professor's intellect was unsettled. This can hardly be admitted, for it will be found on reflection that the complete amnesia of words would produce a like result. This valuable case is strong against the validity of the reading test in cases of aphasia.

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* Papers on Aphasia, already cited.

† Wilbur. Op. cit.

c. The memory of words and signs is an element that is of external origin and artificial formation. Its development varies in different men speaking the same language; that is to say, that some minds seem to have a capacity for a much larger vocabulary than others, the training and education having been similar. There is also a difference between the educated and the ignorant in this respect. Some men (Cardinal Mezzofanti, for example), have acquired the vocabularies of more than seventy tongues. Contrarily, Kasper Hauser, the imprisoned boy of Nüremburg, at the age of sixteen, knew but the words *mann* and *ross*; the only formed stimuli his sensorium had ever received; and yet he was no idiot.* A patient completely aphasic may be made to copy a word, but if the model be withdrawn, it becomes impossible for him to reproduce the combination of letters forming it, the written echo being put down instead. Again, by dint of repetition, the same patient may be made to repeat a word or two, but the matter thus acquired has a very slight hold upon the memory. Even when speech is almost entirely recovered, the affected persons are aware of the remaining blanks in their vocabulary, and will even (Graves) carry about a list of forgotten words to help them out of difficulty. It is stated that Prof. Lordat, after seemingly perfect cure, never could improvise, nor could he speak written lectures from memory; he was forced to have his manuscript constantly before him. That admirable clinical teacher, M. Trousseau (whose premature death is mourned by the profession the world over), taught in his lectures upon the subject that this *amnésie verbale* constituted in some cases the whole of aphasia, in others the greater part.

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* Copland's Medical Dictionary contains an excellent account of him.

d. The willed, co-ordinated movements required for the production of speech, are sometimes entirely wanting in aphasia. The patient makes the attempt to speak and answer, but only succeeds in uttering his inevitable echo. At times, as before stated, words to the point may be forced out by exciting the emotions. Dr. Robertson's patient, when asked what she would do if her shawl were to be snatched from her, exclaimed, to the astonishment of the doctor, "police!" This was repeatedly exemplified in Case III. of this report. Without leaning toward any localization, these movements may be explained as follows: afferent currents, consisting of stimuli (words or signs, *a*) enter by means of the optic, auditory, or general sensory nerves, reaching in all certainty to the thalami optici and the corpora striata; they are according to this hypothesis thence transmitted to the gray matter of the convolution. In this gray matter the necessary purely mental operations (*b*, *c*, and part of *d*) take place, and the willed movements are sent back through the same track, by means of motor nerves, to the organs of articulation, writing and gesticulation. Now, it has been a subject of speculation whether this element (*d*) is impaired in its will part, or whether there existed an interruption in the *conduction*. This last supposition (based upon the fact that the white cerebral matter is almost invariably involved in the lesion), forms Dr. Robertson's* theory of aphasia, he holding that the lesion is essentially a *motor* and not a *mental* one. Prof. Lordat † had such a theory in view when he ascribed the loss of language to an "*asynergie verbale.*" Drs.

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* Robertson. *Journal of Mental Science;* Jan., 1867, p. 503.

† Lordat. *Analyse de la Parole*, etc. Montpellier, 1843.

Letourneau * and Cerise † believe that the condition in question may be due in great part to a lesion of transmission.

e. The production and modulation of sound need not be more mentioned in this connection, as it is evident to any one who has seen a case of aphasia that the patient's larynx and vocal cords perform their functions perfectly. This statement is not intended to apply to such modulations as are required for intricate musical vocalization, though no case has been reported in which laryngeal musical language has been lost, yet such a loss may, *a priori*, be expected to occur.

After this cursory review of the pathology of aphasia, the question of localization presents itself; a question which has recently been discussed in France, more especially, by the most eminent men, and with great vehemence. As the numerous papers on the subject have not done much toward the settling of the vexed points, it will only be necessary to examine the facts brought forward and the arguments used in the two Parisian societies; and as the object of this report is merely to state the progress made in the study of their condition, this examination will be brief and will involve no criticism. The first discussion occurred in the "*Société d'Anthropologie*" in 1861; the second, in the Imperial Academy of Medicine in 1865. For greater precision the two divisions of the question will be treated of separately.

1st. The psychological proposition: there does exist a *faculty of language*, was announced by Gall ‡ in 1809; he following in this the authority of philosophers of the preceding century; it was a part of his celebrated hypothesis, phrenology. It is remarkable that though his doctrine as a whole was immediately attacked, yet this particular proposition was accepted by quite a number of the eminent members of the profession. In 1825, Bouillaud § acknowledged that he recognized a faculty of language, and he brought forward a large number of cases to prove that this faculty was located just as Gall had indicated. The leading medical minds being at that time engaged in the development of new-born pathological anatomy, this psychological aspect of the subject did not receive the attention it deserved. But in the *Société d'Anthropologie* ‖ in 1861, after a rather adverse report by M. Lelut, on a paper by G. Dax, Bouillaud again boldly proclaimed himself a follower of Gall; stating that while he was not prepared to feel bumps, yet he believed the principle of localization of the faculties to be a great truth. This was followed by a powerful debate. Auburtin elaborately supported Bouillaud, while Lelut took strong grounds against phrenology in any form, saying that he was not willing again to take up arms against an error which he had helped to destroy twenty-five years previously. Gratiolet, one of the greatest of French neurologists, followed in a very brilliant and forcible argument, principally of an anatomical nature. He stated that while no positive proofs could be adduced of the non-existence of faculties as distinct, independent portions of mind, yet the very complex nature of these so-called faculties, their mutual connection and dependence, and the observation of the development of the intellect, all tended to show that the mind was a whole, a soul, manifesting itself in protean ways by means of, or through organs. Phrenologists having asserted the affirmative, their opponents were laboring under the disadvantage of having to disprove their proposition. If language could be localized, then the other so-called faculties might as well be, and the human mind would take at once a giant stride into materialism. He concluded with a brilliant protest against the attacks made upon the mental unity of man.

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* L'Union Medicale. 18 Mars, 1865.

† *Journal de Médecine Mentale*, p. 229. Paris, Sept., Oct., 1866.

‡ Récherches sur le Systèmo Nerveux. Paris.

§ Traité de l'Encéphalite. Paris.

‖ Bulletin de la Soc. d'Anthropologie. Tome II., 1861.

In the Academy of Medicine, during 1865,* Bouillaud, powerfully aided by Broca, supported phrenology once more; the debate this time being upon the last and boldest venture of these two distinguished men, viz.: the location of language in the posterior part of the third frontal convolution of the left hemisphere. Trousseau, while citing cases opposed to this hypothesis, certainly seemed inclined to favor it. The leading physicians to the insane, however, Parchappe, Cerise, Baillarger, brought forward many opposing cases and stoutly maintained the unity of the mind. Fournié in his late work,† utterly rejects the idea of the existence of faculties and their localization. Vulpian ‡ has added the weight of his great authority against any such modification of phrenology. In this country, the only writer on the subject, Dr. Wilbur, is opposed to it as well.

2d. The anatomical proposition: As before stated Broca's an-

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* Archives Générales de Méd. 1865, Vols. I and II.

† Physiologie de la Voix et la Parole. Paris, 1866.

‡ Leçons de Physiologie du Système Nerveux. Paris, 1866.

announcement was not, as indeed Bouillaud's and Dax's had been before, by numerous negative cases; and when the last-named discussion took place, carefully made autopsies since 1861 had furnished (Trousseau) fourteen cases for and eighteen against the third convolution view. Various objections were offered to the correctness of these negative examinations, but in the last two years quite a number of others have been made by the most reliable observers, and the cases must be accepted. It will be shorter and clearer to treat of the three localization theories in a semi statistical manner, though it must be acknowledged that the figures given are far from complete, especially those in the negative.

a. Gall's and Bouillaud's localization in the anterior lobes. The number of cases favoring this view has reached (as this class includes the other two) five hundred and fourteen; those against only thirty one,* but this includes four such remarkable instances of injury to the brain without loss of language as to require quotation at length. Professor Bigelow, of Boston, has reported † a case which occurred in the practice of Dr. Harlow, of Cavendish (Vermont). On the 13th of September, 1848, the foreman of a mine, a young and healthy man, was standing over a newly laid blast with a tamping iron in his hands. This was an iron bar, pointed at one end (which end was directed upward), one hundred and ten centimeters in length, three centimetres in diameter, and weighing six kilogrammes. Thinking that the blast had been properly covered with sand, he struck it a blow with the round end of the bar, when a spark flying from the rock ignited the uncovered powder, producing an explosion which drove the tamping-iron completely through his skull. The pointed extremity entered at the angle of the lower jaw, on the left side, passing upward and a little inward, it escaped in the neighborhood of the anterior fontanelle junction. The iron was found, covered with blood and cerebral substance, at a distance of several meters. Shortly after this he sat up and talked while on his way home in a wagon. When seen by Dr. H. the patient was cool and rational, describing the occurrence accurately. The direction of the wound was verified by the introduction of the finger into both openings, and from the place of exit portions of the frontal and parietal bones were removed, leaving an opening nine centimeters in diameter. During the progress of the case the patient retained full command of speech, and though at times drowsy, yet he was always rational. On October 11th, being asked how long had elapsed since the injury, he instantly replied, "Four weeks this afternoon, at half past four o'clock." Recovery was perfect about the end of November, but the sight of the left eye was lost. In January, 1851, he came to Boston and was shown to the medical class by Dr. Bigelow. He was at that time in perfect health. It may be well to mention that this case is supported by the affidavits of a number of reliable and intelligent persons. In the same periodical for January, 1850, is related a case by Dr. Detmold, of New York, in which, after severe injury to the frontal region and secondary suppuration, the anterior left lobe was incised freely twice at least. No difficulty was observed in the speech, and the patient talked a good deal when not comatose. In the debate at the Anthropological Society, Gratiolet detailed the following case from the service of M. Bérard. A man was wounded in the forehead by the explosion of a mine; on being picked up, the patient was rational, and gave an account of the accident. He walked to the hospital, and when seen there by Bérard he had no paralysis and spoke well. Death took place in twenty-five hours, and the autopsy showed both the anterior lobes reduced to a jelly and penetrated by spicula. Trousseau* gives a case no less important observed by M. Peter at the military hospital of the "Gros-Caillon;" that of a cavalryman who, while intoxicated, fell from his horse striking upon the occipital region and fracturing the skull. In the hospital this man developed the wildest delirium, swearing most energetically, and carrying on conversations with imaginary persons. Thirty-six hours later death supervened, and a post-mortem examination showed that the extremity of both anterior lobes had been disorganized by *contre-coup*. The writer has been kindly told† of a case very recently observed by Dr. Stokes, of Dublin, in which, for suicidal purposes, three small balls had been fired from a pistol into the temporal region. The frontal bone was very extensively fractured, the anterior lobes lacerated, and at length nearly destroyed by inflammation, and yet no aphasia manifested itself. Coma came on only in the last days of life.

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* The [illegible] his number might be considerably increased from [illegible] unpublished cases.

† American Journal of the Med. Sciences, July 1850.

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* Clinique Médicale, t. ii. p. 610.

† Verbal Communication, by Dr. Brown-Séquard.

b. The hypothesis of the doctors Dax, father and son (accepted by Bouillaud and Auburtin in 1861), of localization in the *left* anterior lobe. The following table contains the cases that were collected for the purpose of sustaining this view, and the few negative autopsies made recently and more particularly opposed to Broca:

QUESTION OF LEFT ANTERIOR LOBE.

AUTOPSIES BY	FOR.	AGAINST.
Marc Dax (1836) and G. Dax (Acad. de Méd. 1863)	370	—
Bouillaud, 1848	85	—
" 1865	31	—
Trousseau, " (Acad. de Méd.) . . .	18	16
Vulpian (Leçons de Phys. 1866) . . .	5	—
New York Hospital (1830-67)	2	6
Jackson, Richardson, A. Clark, 1866-7 . .	3	—
Peter Legrand, Béclard, Delpech, Bérard, one each	—	5
Farge, Bigelow, Detmold & Stokes (one each)	—	4
Total	514	31

c. Broca's hypothesis of localization in the posterior part of the left third frontal circonvolution, in the neighborhood of the island of Reil. The details of the two cases, and autopsies which gave birth to this idea, and which brought about the "brilliant conversion" of M. Paul Broca to the phrenological doctrine, are to be found in the *Clinique Médicale* of Trousseau, Vol. II., article aphasia. It is useless to reproduce them or any of the twelve or fifteen other cases in which this precise lesion has been found, but for the purpose of guiding future investigations, the following description by Broca, of the topography of the part of the brain involved, may not be without interest.

* "The sulcus of Rolando divides the anterior from the middle lobe; it traverses the external surface of the hemisphere from above downward; starting from the longitudinal fissure to terminate in that of Silvius. Anteriorly this sulcus is bounded by the transverse frontal convolution, posteriorly by the trans-

verse parietal convolution. The anterior lobe thus comprises all that portion of the hemisphere which is (above) anterior to the sulcus of Rolando, and (below) in front of the fissure of Silvius. The inferior part of the anterior lobe is formed by the so-called orbital convolutions. The superior and lateral portions of this same lobe are made up by the proper frontal convolutions. These are three in number: a superior, or first; a middle, or second; and lastly, an inferior one, the third frontal circonvolution. They all run antero-posteriorly, and terminate in the transverse frontal convolutions, of which they seem to be branches. The third frontal convolution is free in its posterior half, and is separated from the middle lobe by the fissure of Silvius, whose anterior border it forms. On account of this relation, the third convolution is sometimes spoken of as the superior marginal, and the first temporo-sphenoidal convolution as the inferior marginal. When these two marginal convolutions are separated, there is perceived, at the bottom of the fissure of Silvius, a rather distinct eminence, from the summit of which start five small convolutions, or more properly speaking, five straight folds, in the shape of a fan; this is the island of Reil, which is directly connected with the extraventricular portion of the corpus striatum."

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* Translated from Trousseau. Op. cit.

The next table contains only such autopsies as have been made with especial reference to this question, or in which the details given were sufficient clearly to indicate the location of the lesion.

QUESTION OF LEFT THIRD FRONTAL CONVOLUTION.

AUTHORITIES.	FOR.	AGAINST.
Trousseau, 1865 (in Acad. de Méd.) . .	14	18
Peter Legrand, Beclard, Delpech, Bérard, Farge, Jackson, Bigelow	—	8
Jackson, Richardson, Russell . . .	3	—
New York Hospital (1830, 1867) . . .	1	7
Bellevue Hospital, Oct., 1867* . . .	—	1
Total	18	34

The following tables, on other points, may prove of interest.

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* Case in service of Dr. A. Clark; mentioned by permission.

1. Aphasia without Paralysis.

Jackson, Lond. Hospital Reports, Vol. I.	10
New York Hospital	2
Total	12

2. Aphasia with Hemiplegia.

AUTHORITIES.	RIGHT HEM.	LEFT HEM.
Trousseau, 1865 (Acad. de Méd.)	125	10
Baillarger, later in 1865 (from Salpêtrière)	30	1
Jackson, loc. cit.	34	3
Robertson, loc. cit.	3	—
Medical Times and Gazette, Sep. 9, 1865.	2	—
Arch. Gen. de Méd. 1866, Vols. I and II.	2	—
Austin Flint, Sen., New York Medical Record Vol. I.	4	—
New York Hospital, 1830–67	43	3
Total	243	17

3. Parts of Encephalon Damaged in Aphasia Cases.

Third Frontal Convolution on left side	19
" " " right side	1
Anterior Lobe of left side	514
" " right side	2
Lateral Ventricles distended	2
Corpora Striata	6
Middle Lobes	3
Posterior Lobes	4
General Softening of one Hemisphere	2
Cases of Aphasia without lesion of brain substance	3
A number of cases caused by reflex action.	

A discussion of the evidence here presented, or the offering of any new theory of aphasia, would be a transgression of the limits of this paper. Besides, this evidence has been deemed, by high authorities, not sufficient to settle the question. Of course, no one will deny that the coincidence of nineteen cases in favor of Broca's view is a most remarkable one, and that it must mean something, if it does not signify that the faculty of language is located in that famous third convolution. To some minds, the cases related, of great injury to the anterior lobes, supported by such names as Bigelow, Gratiolet and Stokes, will at once settle the question in the negative. And many, no doubt, will be inclined to Brown-Séquard's reflex theory, especially if the experience of M. Rufz as to the effect of a poisoned peripheral injury should be confirmed.

All this can only be settled, if at all, by observation. More cases are wanted, and these must be more perfect than any hitherto reported. It is the principal object of the writer to provoke a thorough study of cases by every member of the profession who shall have the opportunity. Publish the cases as soon as complete, and send (if living in the country) the specimens to the pathological society of one of the great cities for presentation. In making the autopsies it is important that the state of the heart and arteries should be ascertained, the branches of the circle of Willis being minutely examined before any incisions are made into the brain. The microscope should be used to discover the state of the histological elements or the presence of pathological ones; and especially, if the third left frontal convolution does not appear altered to the naked eye, ought its substance to be examined with a low objective at the least; and in all cases it is highly desirable that the body should be opened within twelve hours after death, to avoid the occurrence of too great cadaveric change. In carrying on such investigations two things should be kept in mind. Firstly, the condition necessary to the establishment of a theory of aphasia, including localization, as laid down by Fournié: *a.* to find at what part of the encephalon intelligence (the soul) acts upon nerve fibres to excite the movements of speech. *b.* To indicate the seat of perception of the sound—speech. *c.* To determine the anatomical connections by which our perceptions in general act upon the sound—speech, to bring about its reproduction in the sense of hearing, or to provoke the movements peculiar to it (the sound—speech).* And secondly, the warning words with which the late lamented Gratiolet closed his argument in the *Société d'Anthropologie:* "I do not hesitate," he said, "to conclude that all schemes of localization hitherto proposed are without foundation. These are, doubtless, great efforts—Titanic efforts. But when from the top of such a Babel we attempt to seize on Divine truth, the edifice crumbles."

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* Which last proposition evidently refers to internal, unspoken language.

CASES IN WHICH APHASIA OCCURRED WITH RIGHT HEMIPLEGIA.*

I. E. S., æt. 27, seaman, admitted February 10, 1866. The history of case was not fully recorded. On admission, he presented hemiplegia on the left side of body which had lasted four weeks, and of which he was getting better. Speech and intelligence were perfect. February 27.—Yesterday morning, patient remarked that he was nearly well. In afternoon the nurse says he mumbled and talked queerly. This morning he presents complete aphasia. At the visit he was sitting up in the bed looking intelligent but surprised and annoyed at his total inability to find words; this annoyance he expresses by appropriate gestures. There is no paralysis of right side, and yet the power of expressing ideas in writing is lost; cannot even write his own name, Edward. Late to-day, he uttered one word. February 28.—This morning distinct though slight paralysis and anæsthesia of right side of body are found; and there is some difficulty in swallowing. The aphasia is unmistakable, there being no lingual palsy and no impairment of intellect; can't shut his eyes. March 1.—Hemiplegia on right complete save a little motion in leg. Eyes wide open. Ordered iodide of potassium .60 four times daily, and nourishing food. March 6.—Last night spoke a few words quite distinctly; now takes food better. About this time, patient contracted typhus fever and had a pretty smart attack, with a few shallow bed-sores on sacrum. During the height of the fever (treated with infus. serpentariæ, spongings, fresh air and stimulants) the paralysis disappeared in part and his vocabulary increased notably. Though no mention is made of the fact, the writer is confident that the eyelids were closed voluntarily during the intercurrent attack. During convalescence, the motion of the right side became very free, the anæsthesia disappeared, and speech gained daily. April 9.—Note is made that patient sits up; bed-sores are healed; he speaks well and moves limbs freely. Uses faradization, and strych. et ferri citras .12 t.i.d. May 1.—Is discharged well, with the exception of a little embarrassment in motions of right arm.

II. W. H., æt. 33, seaman, admitted February 26, 1867. On the 19th inst., while sailing from Richmond, Va., to this port, a few minutes after eating breakfast, he felt a little dizzy and became aware of loss of power in right half of body; no loss of consciousness; friends did not remark defect in speech. He walked to the ward; the leg had recovered, the grasp of the hand being feeble, however. The face was drawn to the left and there was no anæsthesia. He talked generally as well as men of his class; but on direct questioning, a curious partial amnesia was discovered. He did not recall his name, his age, nor did he remember whence he had sailed, and by which river he came (though he had been in that trade for years). He retained almost all words in common use. It is to be regretted that the power of writing was not tested. On the third day after admission (Feb. 29), he told us his name spontaneously, and not only said he came from Richmond by way of James River, but he was able to speak of the points of interest along that stream, Butler's canal, etc. He had, therefore, completely recovered command over the stock of language used in his sphere of life. As to the probable lesion in this case : he bore no signs of premature decay, and at his age, disease of the vessels of the brain might consequently be thrown out; he had received no injury whatever; but he stated that three years previously he had suffered from sub-acute articular rheumatism. He cannot be made to recall any heart-symptoms, either pain or palpitations. On examination, however, a slight alteration of both sounds of the heart at the base is found. The first sound is very slightly prolonged, and the second has lost its sharp definition. A minute embolism is therefore probable. Urine normal. March 9.—He has had a little strych. et ferri citras, and is to-day discharged, cured.

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* In the period from 1830 to 1867 there were entered in the case-books fifteen cases of right hemiplegia without loss of speech.

III. A. O'B., æt. 28, a single woman, admitted December 28, 1866. Patient has had pretty fair health until the end of November last, when she had an attack of "bilious fever" (?) lasting three weeks. On the morning of Sunday, December 9, being convalescent, while her sister was, 9 A.M., putting clean linen on her, she suddenly exclaimed that she had a pain in the stomach and wanted a drink. In less than five minutes afterward it was noticed that her right side was palsied and that she could say nothing. It seems that she was then unconscious for five or six days, but whether complete coma existed is not certain ; since that sixth day she has been intelligent. From that time her general condition became pretty good ; her appetite increased and digestion was well performed. The sphincters acted normally; the face was drawn to the left; she complained of severe pain in top of head ; a bed-sore formed upon right side of sacrum, and another upon right outer malleolus; and she acquired a few words, "no" and "darling." On admittance, the following points were noted : there is no motion in the right side of the body ; sensibility of skin is not materially affected ; the face is drawn to the left side ; the tongue, when portruded, points to right ; the pupils are normal in size and sensibility. Her intellect seems to be in perfect order ; memory alone being disordered in its relation to language. As concerns the three elements of the functions of language. 1st. Speech proper is substantially lost, all that remains at her command being the words "dear," "darling," "yes," "no," and "I couldn't." Articulation itself is perfect. 2d. It is not possible to ascertain the existence of the power of written language as paralysis affects right hand. 3d. Gesture is completely lost, though her eyes show impatience and grief, yet her left hand and shoulders are not used to express these feelings. There is no palsy of pharynx or of tongue as a whole. Bed sores are improving. Within a week after admission she acquired a few more words, "doctor," "good morning," and "Julia." Great efforts were made by physicians and nurses to teach her by repetition of words and by a sort of objective system, but in vain. Occasionally, new words, chiefly emotional phrases, would be forced from her by joy or sorrow or sudden questioning. She hummed quite a number of tunes as well as before illness. She also began to use the left hand in gesture. January 20, 1867.—She cannot say her own name, though she is angry at being called Brown or Smith; her general vocabulary has not increased. If asked for a thing she points to it or brings it. Her general health is better and the bed-sores have healed rapidly. Face is less distorted, leg moves a little, but arm is useless. The electrical condition of the muscles (sensibility and excitability) is good. Takes iron and strychnia and the iodide of potas., with good food and faradization of muscles. Walks a little; speech same; mind not so bright. Discharged by request.

IV. E.B., æt. 28, seaman, admitted Nov. 23, 1863. After severe vomiting on the 21st, is said to have gradually become hemiplegic on right side. No details of attack. On admission, is deprived of motion on right side; mouth a little drawn to left side; pupils natural; "though patient cannot speak, and cannot or will not make signs, he moves his eyes in a manner that indicates intelligence." November 28.—Since last, three dislocations of jaw have taken place: they were easily reduced. Patient now utters a few simple words, and gains motion. December 26.—Patient now walks tolerably well, and speaks readily. Has had no medicines excepting occasional laxative. Discharged cured.

V. C. O., æt. 50, married woman, admitted July 9, 1864. No history can be obtained from patient. Friends state that four days ago she had an "inward spasm," since which she has been palsied, and has lost her speech. On admission, there is partial paralysis of right side, with slight anæsthesia: mouth is slightly drawn to left side, but tongue protrudes straight. Speech much impaired. Ordered blister to back of neck, and potass. iodide .60 t.i.d. August 18.—Has regained use of limbs, but is merely possessed of a few words. She finds it impossible to express her ideas. Discharged cured.

VI. F. M., æt. 28, seaman, admitted December 10, 1862. On 8th December, paralysis of right side of body, with complete loss of speech came on; no details. On admission, patient is perfectly conscious, and has recovered some speech. There is entire loss of motion on right side: tongue does not deviate. December 15.—Patient got a bronchitis, which to-day proved fatal. No autopsy allowed.

VII. S. F. L., æt. 48, merchant, admitted January 17, 1862. Patient was brought in about midnight, from No. 121 Greene street, where, during the act of coition, he was attacked with palsy. On admission, there exist complete paralysis and anæsthesia of right half of body; tongue deviates to right. Patient appears to understand everything, but cannot answer, excepting by saying "I can't speak," and by signs expressive of despondency. January 20.—Has improved somewhat in speech. Has taken good food, and tinct. aconiti .20 t.i.d. March 4.—Now walks across room pretty well; has still anæsthesia; speaks well, but deliberately. Cured.

VIII. J. H. H., æt. 30, grocer, admitted July 17, 1861. Three years ago patient had an attack of apoplexy (no particulars), from which he slowly recovered, but has retained a feeling of vertigo. Eight days ago second attack came on suddenly, with coma, lasting twenty-four hours. On recovering, it was noticed that he answered slowly, imperfectly, and not to the point. On admission, entire right side is paralyzed, and sphincters relaxed. July 27.—Has some motion in right side, and no longer passes fæces and urine involuntarily; speech the same. Has had a blister between shoulders, and potass. iodide .60 t.i.d. August 24.—Since last, has had specific iritis (had chancres six and eight years ago), and was treated with mercury. Can now walk to window. September 7.—Patient is completely cured.

IX. C. W., æt. 19, single woman, admitted January 15, 1860. Two days ago had attack of paralysis, affecting right side of body and face; "speech is slightly affected." Has had syphilis. May 29.—Under tonics has improved much; walks pretty well, but cannot raise arm. Discharged relieved.

X. A. C. S., æt. 45, merchant, admitted April 20, 1859. Yesterday, while walking, was struck with palsy. On admission, has recovered from coma; is completely paralyzed on right side; recognizes his friends, but cannot protrude tongue or speak. April 23.—Is able to protrude tongue, and says a few words; is perfectly rational. June 16.—Patient has improved in every way under tonics, and is discharged relieved.

XI. D. H., æt. 52, ship-steward, admitted September 1, 1858. Patient was paralyzed in April last. Has been healthy and temperate. Last March had acute rheumatism. On admission, is suffering from loss of power on right side; face paralyzed on left; patient can hardly articulate, and cannot give history; laughs immoderately. September 15.—Symptoms of cerebral softening showed themselves, and have rapidly progressed; died to-day. No autopsy.

XII. P. P., æt. 38, weigher, admitted June 9, 1858. Is brought in after having had a fit. Right side of body, and left side of face are paralyzed. Notes say there is also "paralysis of tongue." June 27.—Discharged at request; not improved.

XIII. Unknown man, admitted October 8, 1857. Brought in comatose. October 9.—After stimulating enemata and sinapisms has rallied. Paralysis of right side, without anæsthesia; is conscious, and understands questions, but makes no reply. October 11.—Died comatose. Autopsy showed left lateral ventricle filled with a recent clot. No details.

XIV. W. D., æt. 65, merchant, admitted 25th March, 1857. Received nearly comatose, with hemiplegia of right side of body and face; left pupil dilated. March 26.—Rallying. "On being questioned, he seems to understand what is said to him, and attempts to answer it. The first two or three words are evidently in reply, the rest incoherent." Urine and fæces passed in bed. April 15.—Died, but no autopsy allowed.

XV. J. H. C., æt. 55, clerk, admitted January 22, 1857. Two hours ago he was seized with hemiplegia of right side. On admission, paralysis is complete, face drawn to left; pupils are natural; "speech mumbling." He appears to understand what is said to him, and tries to answer; has control of sphincters. February 12.—Patient has since had an attack of facial erysipelas, but has steadily improved. Discharged relieved.

XVI. M. W., æt. 16, single, servant girl, admitted September 25, 1856. Patient has never menstruated, but has had palpitations and dyspnœa on exertion, together with tolerably regular monthly epistaxis. Never had rheumatism. Three days ago (after violent headache and lighter epistaxis than usual) her friends noticed that she was palsied on the right side; and in P.M. of same day she became comatose, and remained so until to-day. On admission, patient is conscious; has partial paralysis of right leg, in right arm it is complete; facial palsy on right side; pupils regular. Patient has lost power of articulating words, and merely utters a harsh cry when addressed. A blowing sound is heard all over præcordial space, loudest at apex; impulse of heart is forcibly felt in fifth intercostal space, nine centimeters from median line. Mammæ not developed. October 3.—Patient has improved; face is nearly straight, and to-day she utters a few words. October 10.—Had been gaining steadily, when at 2 P.M. she had a convulsion, became comatose, and at 9 P.M. died. Autopsy, fifteen hours post mortem. Heart weighed five kilos. Aortic valves were healthy; mitral covered with vegetations; brain substance healthy. A clot is found distending the left lateral ventricle, right empty. At base, a more recent large clot is discovered. No mention of state of arteries. A recent corpus luteum found in ovary.

XVII. J. D., æt. 52, omnibus driver, admitted April 9, 1855. While driving, a couple of days ago, was suddenly struck with paralysis. On admission, there is complete loss of motion and sensation on right side of body, and left side of face; patient cannot speak; heart healthy. May 4.—Discharged relieved.

XVIII. F. F., admitted June 29, 1854, in a state of delirium. June 30.—To-day patient is rational, but can give no account of himself, as he can articulate but few words. There is complete paralysis of right arm, and partial of leg; face paralyzed on same side; right pupil larger. July 11.—It is now learned that two months ago patient received a blow on the head, and suddenly became palsied. Is now much better. Eloped relieved.

XIX. E. Van H., admitted May 23, 1854. Has had poor health of late. Yesterday he suddenly lost power of speech, and became paralyzed in right arm. On admission, face is drawn to one side; right hemiplegia; and patient is speechless, but rational. June 1.—Died. Autopsy showed considerable serum under arachnoid, and in ventricles; brain substance considerably softened; no details. Some ascitic dropsy; heart not examined. (This autopsy is not included in tables.)

XX. S. C., æt. 29, seaman, admitted April 4, 1856. In January was suddenly palsied while going from a warm into a cold room. On admission, had a little use of right side; face paralyzed on right side; patient does not speak as plainly as he did before this attack. April 17.—Discharged by request.

XXI. C. W., æt. 33, mechanic, admitted March 13, 1855. On March 9, was found insensible, but recovered in a day or two. On admission, there is paralysis of right side of body and face; sensibility on that side is somewhat impaired; right pupil dilated. March 20.—Examination shows increased dullness over heart, a harsh systolic murmur at apex. Has had rheumatism. June 30.—Has gradually improved. Now walks about grounds, and utterance is much better. July 9.—Discharged cured.

XXII. N. E., æt. 66, seaman, admitted July 16, 1851. Five weeks ago, after lying on deck of a steamship in the sun, he woke with palsy of right side. On admission, right arm is nearly useless, leg not so much affected; no anæsthesia. "Speech is very much affected." Controls sphincters. Ordered tonics and good food. December 1.—Has regained considerable power. May 14, 1852.—Is sent out relieved.

XXIII. Ann B., æt. 55, widow, admitted October 18, 1850. Was well until six months ago, when she began to suffer from severe pain and tenderness in right side of head; some two months later this occupied both sides. Six weeks ago, while in church, she was seized with a general palsy. Her consciousness did not leave her, but she lost the power of speech and motion; she cannot say for how long a time. Has since had two similar attacks of same nature. Denies having had syphilis, but she has had much rheumatism in long bones. On admission, no mention in notes of paralysis or loss of speech. Was leeched and salivated. February 6, 1851.—Has greatly improved; hardly any pain. April 2.—Sent away disorderly.

XXIV. Ellen M., æt. 26, single, cook, admitted January 20, 1850. One week ago, while crossing a street, she fell down in an apoplectic (?) attack, but was soon able to get up, walk home, and resume work. Three days since she was troubled with numbness, and then had a seizure like the first, which left her with right hemiplegia. On admission, there is complete palsy of right side; patient unable to speak. January 22.—Heart healthy; still can't speak. Leeches to temples and tartar emetic *ad nauseam*. February 1.—A little motion in right leg, and she speaks a little. Of late has had strychnia .008 bis die, with electricity to affected limbs. Discharged relieved.

XXV. M. T., æt. 30, seaman, admitted June 6, 1849. Three days ago was struck on head by a falling mast. On admission is paralyzed on right side, but is wide awake. Is obstinately constipated. At last (after many other medcines), four drops of croton oil have produced a moderate stool. Ordered calomel to affect system. Nov. 12.—After mercurial course he improved until middle of July; since no change; "talks incoherently." Discharged.

XXVI. C. L., æt. 60, admitted August 6, 1848. Has just come from Europe in steamer. Is hemiplegic on right side; is very stupid, but not delirious. Aug. 15.—Is not as strong as on admission. Is unable to articulate with any distinctness. 27.—Died. Autopsy, ten hours post-mortem, revealed a cyst containing 65.cc of serum in right hemisphere over ventricle, but unconnected with it. Other organs not examined.

XXVII. A. L., æt. 40, civil engineer, admitted April 12, 1848. Has been a pretty hard drinker for two or three years. Four days ago had an apoplectic fit with coma for half an hour. On admission, complete paralysis and anæsthesia of right side; all power of articulation lost; ordered strychnine .004 t.i.d. June 13.—For a month has been able to walk in garden with a cane; speaks more distinctly. Discharged relieved.

XXVIII. A. B., æt. 70, seaman, admitted April 9, 1848. Four days ago had a sudden loss of power on right side; has had several such attacks before. On admission, there are paralysis and anæsthesia of right side; patient is obliged to articulate slowly. 15.—In spite of electricity and strychnia he died to-day. Autopsy ten hours after death showed serous effusion under arachnoid and in left ventricle; much softening of left hemisphere.

XXIX. P. B., æt. 17, clerk, admitted Sept. 17, 1847; two weeks ago was noticed to be dull; one week ago, after a fit, was found to be palsied. On admission, has paralysis (no anæsthesia) of right side of body and face; speech is muttering and unintelligible; face rather stupid. Dec. 4.—Has had tonics, strychnia and electricity; can use arm and leg; no mention of state of speech. Discharged cured.

XXX. P. N., æt. 52, shoemaker, admitted May 22, 1846. Six weeks ago, after a fit of anger, patient had an attack of apoplexy. On admission, right side of body is recovering from paralysis ; patient "speaks with difficulty." June 19.—Now walks in yard ; discharged relieved.

XXXI. C. R., æt. 53, seaman, admitted February 21, 1846. For a month has had pain in back of head; on trying to rise, ten mornings ago, he found his right side paralyzed and speaking difficult. 25.—Feels better than on admission ; heart is a little large, no murmurs ; has some pain over left lobe of cerebellum ; does not speak as well as usual ; no fever. March 2.—Has lost power of speech almost entirely ; the left side of body is now paralyzed more than right, which still improves. 4.—Walks a little and speaks better ; has been cupped, and had low diet. May 7.—Has much improved ; speaks fairly ; electricity daily applied. July 16.—Discharged relieved.

XXXII. Mary V., æt. 56, married, admitted July 14, 1846. Four weeks ago, after a fall on left side of head, she became unconscious ; on coming to, was found hemiplegic on right side. On admission is paralyzed on right side of body ; tongue points to left ; "she cannot give a good account of herself." 23.—Is discharged as improper object.

XXXIII. John H., admitted Nov. 27, 1845. Yesterday was attacked with apoplexy. On recovering consciousness he was unable to open his mouth or to articulate. Right side of body partially, and left side of face completely paralyzed. 29.—Seems better ; jaws relaxed ; swallows more easily. Dec. 4.—Has gained some power over side, but is still unable to speak. Discharged by request.

XXXIV. F. R., æt. 22, fireman, admitted May 9, 1845. Five weeks ago patient fell from a locomotive, striking his head violently, but from this he apparently recovered. Ten days ago, while seated, he suddenly fell, and since his right side has been entirely paralyzed, and his speech rather inarticulate ; no anæsthesia ; the sight of right eye is impaired, and its pupil does not contract well ; the tongue goes to the right, and the right side of face is red and hot, as is also the conjunctiva of same side ; this phenomenon is exactly limited by median line. Nov. 17.—Has had electricity and tonics ; is much better ; can speak more distinctly. Discharged, improper object.

XXXV. Mary C., æt. 47, widow, admitted Oct. 26, 1844. Six days ago she went to bed feeling as usual. On awaking found right side drawn up and paralyzed ; in course of day lost all power of motion, and vision became dim; no coma. On admission there appears to be a little anæsthesia on right side; tongue goes to right ; speech affected. Nov. 6.—Last night had a slight convulsion ; was leeched and cupped with relief. Nov. 22.—Patient is discharged, cured.

XXXVI. A. W., æt. 20, male, admitted Dec. 20, 1844. Patient remained in the ward until Dec. 26 in a semi-comatose state ; without palsy, except that tongue pointed to right side, and that when spoken to he only answered "no ;" on this day he walked about and went to bed in afternoon better ; was found on 27th, in morning, with complete paralysis of right ride. 30.—Lies quietly ; is a little more lively, but still says "no" to everything. Jan. 23, 1845, is not better. Discharged, improper object.

XXXVII. W. F., æt. 40, seaman, admitted March 7, 1845. A few days ago, being in apparently good health, he suddenly had a convulsion, followed by two others, and by complete paralysis of right side with inability to speak; on admission, cannot speak at all; moves leg a little, but arm is motionless and anæsthetic; ordered blisters to back of neck and electricity. May 5.—Has taken strychnine with no benefit; now walks about, but can't speak. June 26.—Uses a very few words; otherwise no change. Discharged by request.

XXXVIII. G. B., æt. 45, admitted July 14th, 1843. Patient has been suffering from rheumatism for some weeks. About ten days ago he suddenly fell down in an apoplectic seizure; has not uttered a word since. His right side is entirely paralyzed. July 22.—Has been getting feebler. Died; no autopsy allowed.

XXXIX. N. G., æt. 39, seaman, admitted April 12th, 1842. Patient has been ill over one year. At that time had an apoplectic stroke, followed by right hemiplegia and loss of speech, from which he has gradually recovered. Is received for obscure disease (cellulitis) of right lower extremity. Had violent hiccough and gradual failure. April 13th.—Died. Autopsy, nine hours after death, showed cerebral substance firm and natural. In left corpus striatum, a cavity the size of a large pea, containing fluid; none in ventricles. Leg was enormously swollen, dark and covered with gangrenous bullæ. No obstruction found in arteries or veins; cellular tissue and muscles easily torn; no heart or kidney lesions.

XL. J. Y., æt. 28, tailor, admitted May 18th, 1843. Six weeks ago had epistaxis, which lasted three weeks, (!) and upon this ceasing while in bed, he had an apoplectic stroke, losing motion and sensation in right half of body; speech completely lost; within two days has spoken a little. June 1.—Is not much improved. Discharged; improper object.

XLI. Mary M., æt. 49, married, admitted January 14th, 1843. Had apoplexy on January 12th. On admission, has paralysis without anæsthesia, on right side; mouth is drawn to left. Seems to comprehend questions, but speaks in a hesitating manner. Discharged February 15th, by request.

XLII. Oliver R., æt. 36, seaman, admitted December 25, 1818, for hemiplegia. A short time before he was suddenly seized with a pain in left side of head, and fell down in a fit. He recovered from this but gradually, having lost the use of the muscles of the right side of face (body meant); mouth and tongue are drawn to left side; right eye has photophobia; articulation is much disturbed. Has equal command over upper and lower extremities. Was purged actively. On 19th of December, while at dinner, he suddenly had vomiting; sweated profusely in evening and complained of cold. At night he became comatose. December 20.—Has been bled without effect; died. Autopsy; great serous effusion under arachnoid. About middle of the anterior lobe of left hemisphere an abscess was found, which extended externally to the dura mater, and appeared internally connected with the lateral ventricle. It was about four inches in circumference, and contained a dirty colored fluid. The surrounding brain substance was much diseased and broken down. A portion of the cerebellum was indurated, the dura mater covering it appearing to have been inflamed. Thoracic and abdominal viscera normal.

XLIII. A. F., æt. 48, painter, admitted September 15th, 1848. One week ago was suddenly affected with loss of sensation and motion in right side of body; "a great impediment in his speech soon followed." On admission is as above; left pupil dilated. September 29th.—Discharged relieved. This patient was seen later, when he had completely recovered.

CASES IN WHICH APHASIA OCCURRED WITH LEFT HEMIPLEGIA.

XLIV. J. J., æt. 40, seaman, admitted June 9, 1850. Has been working in fire room of steamship at night; on 5th inst., after having headache several days, he went in evening to water-closet, and, while there, had a severe pain in shoulder, and pricking sensation over entire right side; found that he had lost power over left. On admission has complete paralysis, and slight anæsthesia of left side of body, "utterance is imperfect." October 15th.—Has had tonics and electricity; is discharged cured.

XLV. J. B. M., æt. 38, book-keeper, admitted Sept. 25, 1855. On 22d., while going to dinner, he suddenly fell; he did not lose consciousness, and had no convulsions. On trying to rise, he found his left side paralyzed, and was unable to call for assistance; next morning was found and taken care of. On admission, the left side is totally palsied, but not anæsthetic; face turned somewhat to one side; "has some slowness and difficulty of speech." He seems to have enjoyed good health previously, and had been regular in habits; may have had occasional headache. October 18th.—Is discharged, relieved.

XLVI. J. S., æt. 35, soldier of 61st Regiment, N. Y. volunteers, admitted September 20, 1861. Patient was transferred from State Hospital; has been hemiplegic on left side, and has lost speech; is now able to walk about and help himself. No treatment excepting good food. January 20, 1863.—Patient is transferred to United States Army General Hospital in this city.

CASES IN WHICH APHASIA OCCURRED WITH DOUBLE HEMIPLEGIA.

XLVII. J. R., æt. 62, porter, admitted December 6, 1860. Brought in semi-comatose, with left pupil dilated and fixed; face flushed; pulse irregular, body relaxed. December 9. Patient is rather more conscious; tries to protrude tongue when asked; left side of body is completely paralyzed, right partially so. Ordered wet cups to temple and pot. iod., .60 t.i.d. 12th. Patient now moves right side pretty freely and left slightly. He understands all that is said to him, but cannot speak. Takes beef-tea and pot. iodid. January 7. After last note patient got bad bed-sores, and last week was attacked with facial erysipelas, in spite of supporting treatment, he died to-day. No autopsy.

XLVIII. R., æt. 41, seaman, admitted September 2d, 1841. About one month ago while lying in berth, he suddenly became paralyzed on both sides at the same moment. Since, he thinks that he has gained some power over legs. September 11. Has been taking iod. potass. Last night he was attacked with spasmodic twitching of all his limbs and right side of face.

Cannot swallow as well as before, and his speech has entirely failed. He appears to understand, and attempts to speak. Tongue is protruded; sphincters paralyzed. Cont. iod. potass. September 27.—Has had a good deal of rigidity of limbs; and has gradually failed; died. Autopsy, eight hours post mortem, head only examined. The superficial vessels are congested; some 65. cc. of limpid fluid in cells of pia mater. The arachnoid at base was cloudy and tough. When transverse sections of the brain were made, it was found to be of a natural consistency. The medullary portion was covered with numerous points of blood; nothing peculiar was discovered in hemispheres. The ventricles contained about 15. cc. of serous fluid. The right corpus striatum was found diseased. Its cineritious portion for the space of a hickory nut (2. centimeters in diameter), was transformed into a soft, reddish vascular pulp. The left was healthy, as were theoptic thalami. The rest of the brain was healthy.*

CASES IN WHICH APHASIA OCCURRED WITHOUT HEMIPLEGIA.

XLIX. T. R., æt. 35, seaman, admitted June 14, 1860. Patient has always been healthy; somewhat intemperate; never had syphilis. This afternoon, while working, he fell down unconscious. On admission, is insensible; had no convulsions; pulse 90; no paralysis; heart healthy. June 15.—Has slowly returned to consciousness. There is a complete loss of speech; "muscles of his tongue and of speech are entirely paralyzed, so that he cannot protrude his tongue nor utter a syllable. His intellect appears to be slow, though he evidently comprehends everything." Answers by signs of head. Is able to walk about; eats and sleeps well. June 30.—Continues to improve; is slowly regaining speech, but cannot protrude tongue. July 13.—Patient now protrudes tongue well, and speech is slightly less difficult. Discharged relieved. (If the patient spoke at all, his mutism must have been owing to something besides tongue paralysis.)

L. K., æt. 21, seaman, admitted November 14, 1866. Patient gave an uncertain history of intermittent fever, contracted a month or so before in one of the Gulf ports. On admission, he spoke in a peculiar manner. The first words of any sentence came forth easily and naturally, but the last, often including the important verb or noun, were either gotten out with difficulty, or not at all. The articulation was perfect, but there was a notable failure of memory of which he seemed aware. He had no headache, and no sign of paralysis. He presented the appearance, so commonly seen in patients coming from the South, designated as malarial cachexia. November 15.—Patient seems to have fallen into a curious lethargic state. The speech is almost entirely lost; but there is neither delirium nor coma; he is intelligent, understanding and answering gestures; he makes use of a few words, but never to the point; he seems to wish to be let alone; urine and fæces passed spontaneously, and are normal; the symptoms do not resemble those of congestive malarial fever; the eyes are not remarkably brilliant, and he does not seem to have headache; the pulse ranges about 100; nothing abnormal heard in heart or lungs; is taking .75 of quiniæ sulphas under the skin, daily, in three doses; whiskey 16. cc. and beef-tea. 19.—In the last few days there has been no marked change in his condition; he often makes use of the phrase "what is it?" sometimes says "yes" without reason; still seems rational; puts out his tongue by imitation and quickly too; has been restless, but at no time delirious; two or three times a day he makes use of a few other words; once yesterday saying five in answer to a question, but not forming any sentence; he seems to have rather more fever, with but small morning remissions; bowels being constipated, are to-day moved by injection; when he has a call of nature he demands the nurse's help with the phrase "what is it?" Cont. quiniæ and food; seems to be failing in spite of this; tongue coated, but never dry. 23.—Fever seems to have assumed a more continued type; no change in cerebral symptoms; no paralysis; no delirium; speech same; has had infusion of serpentaria instead of quinia lately. 24.—Rather suddenly, at 4 o'clock A.M., died.

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* These cases are open to the objection that they might have been instances of complete paralysis of the tongue. But in case XLVII., no difficulty in swallowing is mentioned; and in case XLVIII., it is positively stated that the tongue was protruded. Dr. Jackson's opinion on the bearing of such an objection to cases of aphasia is worth reproducing. "I may say, too, that I have never seen paralysis of the tongue (*i.e.* decided paralysis. . . .), even on one side, with pure hemiplegia. There is, generally, weakness, affecting all the four limbs, although the limbs on one side may be weaker than on the other. I state this as a mere matter of fact, but it is easy to explain it by anatomy. The nuclei of the ninth nerves are imbedded very near the decussation of the fibres from the limb, so that an injury involving the nucleus even of one side, would be, from continuity, likely to damage the motor fibres of both sides of the body—those on one side before, and those on the other after they had crossed." And he adds in another place, that dumbness (paralysis of the tongue) would always be accompanied by difficulty in deglutition or by the impossibility of the act.

The autopsy, nine hours after death, was made with great care. On removing the calvarium, the dura mater was found much congested, the arachnoid remarkably dry, and the vessels on the convexity of the brain greatly distended with blood; not a trace of lymph was here found. On examining the base of the brain, however, a large patch of false membrane was found extending from the optic commissure backwards over the crura cerebri, the pons Varolii and the medulla oblongata, terminating in a thin film on the inferior surface of the cerebellum. The membrane was thickest over the pons, being there 2.5 millimeters through and very firm. None of the nerves appeared altered, nor was there any change in the brain substance beneath the membrane. Anterior lobes (particularly the left with the third convolution and island of Reil) were examined with minute care for evidences of softening, inflammation, hemorrhage or plugging of vessels, but nothing could be found. Horizontal section showed considerable punctate injection. The lateral ventricles were about half filled with a clear, straw-colored serum. The whole encephalon was then finely sliced, but no farther lesions were discovered. Lungs, spleen, liver and kidneys were congested. Heart was examined, but condition is not stated in notes.

The results in these fifty cases were as follows: died, 14 (9 autopsies); cured 11; relieved 13; not relieved, 12; total 50.

CASE OF TRICHINOSIS.*

J. B., a grocer's clerk, aged nineteen years, was admitted into the New York Hospital, service of Dr. Thomas F. Cock, June 19, 1867, suffering from anasarca. The patient stated that he had been ill three weeks, and that during the first ten days he had suffered from violent vomiting and diarrhœa, followed by great debility and by dropsy. On admission he appeared extremely anæmic ; the œdema was so remarkably elastic that pitting could only be produced upon the legs ; the tongue was clean, smooth, devoid of epithelium, and in its anterior part disposed to dryness ; the stomach was irritable, he said, but he managed to retain at least one meal a day ; during the past few days constipation had succeeded to the previously relaxed state of the bowels ; the pulse was weak, small, and beating about 112 times a minute ; respiration was normal, and there was no increase of bodily temperature. The urine was at once examined, and found to be of a pale color, of normal specific gravity, and perfectly free from albumen. The patient was ordered 120. cc. of sherry wine, milk diet, and a small dose of the ammonio-citrate of iron in tincture of cinchona. My suspicions that the case was one of trichinosis were strengthened by the negative results of the urinary examination. Direct questioning elicited the fact that considerable muscular pain and soreness had been experienced during the second and third weeks of illness ; and that previously to the attack he had been in the habit of eating half-cooked ham in sandwiches, and also of occasionally cutting off small pieces of raw ham in the grocery and eating them.

On the 28th the patient had gained a little strength, vomiting having occurred a few times. Eggs and milk had been eaten, and 180. of wine drunk daily. The tartrate of potassa and iron had been substituted for the citrate, as a chalybeate less likely to disturb the stomach. Repeated examinations of the urine had been made with uniformly negative results. On stating to Dr. Cock my reasons for venturing a diagnosis of trichinosis, he very kindly gave permission to resort to the proving examination. The patient readily and intelligently gave his consent. Localized anæsthesia was produced by means of ether and Richardson's apparatus, and a small portion of the deltoid muscle was removed. On tearing apart the muscular fibres in diluted glycerine and looking at the preparation with a low objective, ten or twelve living trichinæ were seen. The cysts were very distinct, located in the muscular substance, their perfectly limited rounded ends beginning to become opaque from granular deposit. About their extremities were masses of fat globes and cells.

The trichinæ which were still inclosed in cysts presented a constant vermicular motion; while those which were free in the liquid of the preparation were so active as at times to leave the field (a narrow one at one jump. These movements were retained by the trichinæ, thus prepared, for four days.

It was Dr. Cock's opinion, that as all symptoms of intestinal irritation had ceased, and as it might be presumed that the parasites had all reached a muscular lodgment, there were no indications in the case, excepting to feed the patient carefully and to give him iron cautiously. This plan was carried out, and slow improvement was observed daily in color and strength; the swelling disappearing rapidly from all parts excepting the legs. August 13.—The patient, although still pale, is discharged cured.

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* From the *New York Medical Journal*, May, 1868. This was probably the first case of trichinosis in America where the diagnosis was confirmed during life by the microscope. The case occurred while Dr. Seguin was House Physician of the Hospital.—R. W. A.

It is to be regretted that the duties of the hospital were so pressing upon the resident staff as to prevent the proper inquiries and examinations so essential to the completeness of such a case.

A CASE OF ACUTE OCULAR ŒDEMA—CAUSE UNKNOWN.*

B., a private in Co. B, 3d U. S. Cavalry, aged 18 years, a native of Pennsylvania. Has never enjoyed good health. From the age of 15 to date of enlistment, last March, he worked in an iron mill, and was exposed to great heat and vivid light. A few years ago he had intermittent fever. Has always had a tendency to diarrhœa, and is now under treatment for an attack that threatens to become chronic. He is pale, flabby, looks old, face and forehead wrinkled. When a child he suffered from night blindness. Sept. 24th, he presented himself with his *left* eye much swollen; says that ten minutes before his eye was well. He was reading, when suddenly a severe smarting pain was felt in the inner canthus, and was at once followed by swelling. He is certain that no foreign body entered the eye. The lids are externally œdematous and nearly closed. On opening them, an almost complete chemosis is seen, the ring being imperfect at its upper outer part. There is no injection of bloodvessels and no lachrymation; pupil normal. I applied a solution of nitrate of silver (3 to 30. aq) freely over the chemosis, and directed a light wet compress to be applied during the evening and night. The next day the eye was about normal.

Second attack, Oct. 3d, at 1 P.M. I had occasion to see a wounded man in my hospital, and asked several questions of B., who was watching him. His eyes were then in a normal state. Hardly had I returned to my room—certainly not more than five minutes than B. came to me hurriedly with his *right* eye swollen. He had experienced the smarting, and the appearance of the eye was precisely similar to that of the left eye ten days previously. There was no sign of inflammation. At 2 P.M. a complete and heavy ring of swelling surrounded and nearly buried the cornea. I pursued the same plan of treatment as before, and in twenty-four hours all was well.

Patient states that he had the first attack of this curious affection in 1860, when at work in [illegible]. Since he has had from two to six attacks each year. [illegible] he was affected once in April and once in July, besides [illegible] above. He [illegible] that in all instances the [illegible] have been the same, and often the disease subsided [illegible] both eyes were affected at one time.

[illegible] have suffered

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* [illegible] N. Y. Medical Record, Jan. [illegible]

from repeated similar attacks of œdema of the eyes. His father has excellent sight, though he has worked for many years in the iron mill.

The irides are pale gray ; pupils habitually a trifle dilated ; urine contains no albumen ; heart healthy. Patient suffers much from epistaxis, in summer mostly.

There are three main points of interest in this case. *First*, the extremely sudden formation of the œdema and absence of the usual symptoms attending inflammatory processes.

Second, the apparent hereditary character.

Third, the coincidence of the disease with a semi-cachectic state.

CAS DE LÉSION PROBABLE DE LA MOITIÉ LATÉRALE DROITE DE LA MOELLE ÉPINIÈRE, DANS LA RÉGION CERVICALE INFÉRIEURE, AYANT PRODUIT DE L'ANESTHÉSIE D'UN COTÉ ET DE LA PARALYSIE DE L'AUTRE.*

Le sujet de cette observation, le capitaine R., du 3e régiment de cavalerie, dans l'armée des États-Unis, âgé de 36 ans, naturellement très-fortement constitué, a toujours joui d'une santé exceptionnellement bonne. Étant en garnison à Natchez, dans l'État du Mississipi, pendant l'automne de 1863, il lui arriva un accident qu'il décrit en ces termes : Il était dans l'habitude chaque jour de se promener à cheval. Le 21 octobre, prenant cet exercice, il aperçut une rue dans laquelle il n'était jamais entré, et s'y lança au grand galop. A peine y fut-il entré qu'il vit que c'était une impasse. M. R. ne put arrêter son cheval, et tous deux furent précipités dans un fossé ayant des murs de briques et d'environ 15 pieds de largeur et 12 de profondeur. Ce fossé terminait l'impasse, et de l'autre côté se trouvait une maison.

Malgré la force terrible du choc, le cheval ne fut nullement blessé, mais M. R. fut relevé insensible et porté à l'hôpital militaire.

Environ une heure après l'accident, il revint à lui et constata sa condition. Il n'avait qu'une seule blessure, une lacération du cuir chevelu sur le pariétal gauche, mais son corps était si complétement paralysé à droite qu'il ne pouvait même remuer un seul doigt. Dans ce même côté la sensibilité était conservée, et il pense même que les sensations tactiles étaient plus distinctes qu'à l'état normal. Dans le côté gauche du corps il y avait anesthésie incomplète, avec conservation du mouvement volontaire. La perte de sensibilité était totale dans les parties animées par le nerf cubital. La tête était fortement inclinée vers l'épaule droite. Il est certain qu'il n'y avait point de paralysie à la face, et que ses sens étaient normaux. Pas de paralysie des sphincters ni d'anaphrodisie. Le peu de dyspnée qu'il ressentit les premiers jours se dissipait lorsque ses épaules étaient soulevées. Il ne perdit plus connaissance et n'eut pas de délire.

Le troisième jour, le mouvement volontaire lui revint un peu dans le côté droit, et après cela il s'améliora progressivement. Vers la fin de la deuxième semaine il quitta son lit, avec l'aide du garde-malade, et fit quelques pas dans la salle en s'appuyant sur le dos d'une chaise ou sur le bras du garde-malade. Une semaine plus tard il se promenait lentement, faisant peu de pas à la fois, tout seul. A cette époque (troisième semaine), il s'aperçut pour la première fois que la sensibilité des doigts de la main gauche s'améliorait, mais il ne remarqua pas l'état de la jambe. En sortant du lit, il fit l'observation que le côté droit du corps se refroidissait plus facilement que le gauche, particularité qui persiste encore aujourd'hui. Six mois après l'accident, il se rendit à son régiment, et avec de l'assistance en montant, put aller à cheval. A cette époque, le cou s'était complétement redressé, et depuis lors il a continué à gagner lentement en force et en agilité du côté droit.

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* Extrait de l'Archives de Physiologie normale et pathologique, mars, 1870.

Au moment où cette observation est rédigée (juin, 1869), M. R. est en parfaite santé et soutient très-bien les fatigues de la guerre. A pied il a l'air un peu gêné, mais cela tient à un degré très-minime d'atrophie du côté droit du corps. Une fois à cheval, il est complétement dans son élément et se montre admirable cavalier. Sa force physique est grande, et il fait l'exercice du sabre également bien des deux mains ; la main droite est un peu congestionée, et les doigts sont en apparence contracturés, mais cela est plutôt une attitude vicieuse qu'une rétraction musculaire. Avant son accident, il écrivait de la main droite ; maintenant il conserve l'habitude prise pendant les mois de paralysie d'écrire avec la gauche, mais il signe toujours avec la droite. La jambe et la cuisse gauche n'ont pas une sensibilité tout à fait normale, autant que je l'ai pu estimer avec une épingle. Il pense que la main droite est encore hypéresthétique, et il a remarqué que de ce côté les ongles poussaient plus rapidement qu'à gauche.

L'histoire du traitement est plutôt curieuse qu'importante. Les chirurgiens à l'hôpital militaire de Natchez crurent à une lésion dans l'hémisphère cérébral gauche. L'erreur de diagnostic s'explique en partie par la présence des deux faits que voici : plaie sur le pariétal gauche et hémiplégie à droite. Ils décidèrent de laisser M. R. sans traitement, sauf qu'après les premiers jours le galvanisme (courant électro-magnétique interrompu) fut appliqué journellement aux membres paralysés pendant quatre mois. Dans le mois d'octobre 1864, profitant d'un congé, il se mit entre les mains d'un horloger de Louisville, qui lui fit des applications irritatrices produisant des pustules. Il se servait d'un appareil composé d'une douzaine de pointes d'aiguilles, avec lequel il faisait pénétrer une pommade dans la peau du côté droit, qui était encore faible. Toutes les parties de ce côté du corps furent l'une après l'autre couvertes de pustules, le traitement durant plusieurs semaines. M. R. croit que cette médication lui fit du bien, et depuis il ne s'est plus soigné.

Cette observation est malheureusement très-incomplète. Elle repose presque entièrement sur ce que le malade a lui-même recueilli. Je n'ai pu étudier d'une manière satisfaisante l'état des diverses espèces de sensibilité dans le côté gauche, car les détails du cas me furent communiqués pendant une expédition contre les Indiens dans le territoire du Nouveau-Mexique, et ce n'est pas en pareilles circonstances qu'un médecin se trouve muni d'æsthésiomètre, etc.

La première question qui se présente en cherchant à analyser le cas est quelle partie du système nerveux central a été lésée.

On peut complétement rejeter l'opinion des chirurgiens qui traitèrent

M. B. en premier lieu. A moins de supposer deux lésions simultanées, une dans l'hémisphère gauche et l'autre dans la moelle épinière, les symptômes ne peuvent être expliqués. Mais l'absence de paralysie faciale s'oppose à l'admission de l'existence d'une lésion cérébrale. Le malade a souffert des symptômes qui suivent :

A droite.	*A gauche.*
1. — Paralysie complète du mouvement volontaire dans les deux extrémités.	1. — Conservation du mouvement volontaire.
2. — Une contracture (?) de muscles inclinant la tête vers l'épaule.	2. — Une paralysie (?) des muscles dont l'action est d'incliner la tête vers l'épaule.
3. — Une hypéresthésie dans ces mêmes extrémités (?). Certainement conservation de sensibilité.	3. — Anesthésie complète dans la région animée par le nerf cubital, incomplète ailleurs.
4. — Congestion de la peau et développement des ongles plus rapidement que du côté opposé.	

On ne peut s'empêcher de reconnaître ici les signes d'une hémiplégie spinale ; une forme de paralysie dont on doit la caractérisation à M. Brown-Séquard.[*]

Se basant sur une physiologie de la moelle épinière qu'il avait lui-même, par de remarquables découvertes, plus contribué que personne à établir, il parvint à expliquer les résultats, en apparence si obscurs et indéchiffrables, de certaines lésions spinales. M. Brown-Séquard a récemment[†] publié une série de leçons sur l'hémiplégie spinale, remplie de faits nouveaux qui ne laissent plus de doutes sur le sujet.

En comparant les symptômes présentés par le capitaine R. avec ceux des malades étudiés par M. Brown-Séquard, il est certain qu'ils appartiennent à une même catégorie. La lésion dans l'observation que je rapporte était donc dans la moitié lateral droite de la moelle épinière.

A quelle hauteur siégeait la lésion? Il est certain que les nerfs diaphragmatiques n'ont point été paralysés, car le peu de dyspnée dont il s'est plaint se dissipait immédiatement après l'élévation des épaules. La sensibilité était le plus affectée dans les parties où se distribue le nerf cubital, nerf qui semble prendre son origine des deux cordons inférieurs du plexus brachial, cordons qui sont formés par la dernière racine cervicale et la première dorsale. Conséquemment la moelle n'était point lésée au niveau de la quatrième paire cervicale, mais l'état pathologique était à son maximum à l'origine de la huitième paire.

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[*] Lectures on the physiology and pathology of the nervous centers : delivered before the Royal College of Surgeons of London in May, 1858.—Philadelphia, 1860.

[†] *Lancet*, Nov. 7, 1868, et dans plusieurs numéros suivants.

XXX. P. N., æt. 52, shoemaker, admitted May 22, 1846. Six weeks ago, after a fit of anger, patient had an attack of apoplexy. On admission, right side of body is recovering from paralysis; patient "speaks with difficulty." June 19.—Now walks in yard; discharged relieved.

XXXI. C. R., æt. 53, seaman, admitted February 21, 1846. For a month has had pain in back of head; on trying to rise, ten mornings ago, he found his right side paralyzed and speaking difficult. 25.—Feels better than on admission; heart is a little large, no murmurs; has some pain over left lobe of cerebellum; does not speak as well as usual; no fever. March 2.—Has lost power of speech almost entirely; the left side of body is now paralyzed more than right, which still improves. 4.—Walks a little and speaks better; has been cupped, and had low diet. May 7.—Has much improved; speaks fairly; electricity daily applied. July 16.—Discharged relieved.

XXXII. Mary V., æt. 56, married, admitted July 14, 1846. Four weeks ago, after a fall on left side of head, she became unconscious; on coming to, was found hemiplegic on right side. On admission is paralyzed on right side of body; tongue points to left; "she cannot give a good account of herself." 23.—Is discharged as improper object.

XXXIII. John H., admitted Nov. 27, 1845. Yesterday was attacked with apoplexy. On recovering consciousness he was unable to open his mouth or to articulate. Right side of body partially, and left side of face completely paralyzed. 29.—Seems better; jaws relaxed; swallows more easily. Dec. 4.—Has gained some power over side, but is still unable to speak. Discharged by request.

XXXIV. F. R., æt. 22, fireman, admitted May 9, 1845. Five weeks ago patient fell from a locomotive, striking his head violently, but from this he apparently recovered. Ten days ago, while seated, he suddenly fell, and since his right side has been entirely paralyzed, and his speech rather inarticulate; no anæsthesia; the sight of right eye is impaired, and its pupil does not contract well; the tongue goes to the right, and the right side of face is red and hot, as is also the conjunctiva of same side; this phenomenon is exactly limited by median line. Nov. 17.—Has had electricity and tonics; is much better; can speak more distinctly. Discharged, improper object.

XXXV. Mary C., æt. 47, widow, admitted Oct. 26, 1844. Six days ago she went to bed feeling as usual. On awaking found right side drawn up and paralyzed; in course of day lost all power of motion, and vision became dim; no coma. On admission there appears to be a little anæsthesia on right side; tongue goes to right; speech affected. Nov. 6.—Last night had a slight convulsion; was leeched and cupped with relief. Nov. 22.—Patient is discharged, cured.

XXXVI. A. W., æt. 20, male, admitted Dec. 20, 1844. Patient remained in the ward until Dec. 26 in a semi-comatose state; without palsy, except that tongue pointed to right side, and that when spoken to he only answered "no;" on this day he walked about and went to bed in afternoon better; was found on 27th, in morning, with complete paralysis of right ride. 30.—Lies quietly; is a little more lively, but still says "no" to everything. Jan. 23, 1845, is not better. Discharged, improper object.

XXXVII. W. F., æt. 40, seaman, admitted March 7, 1845. A few days ago, being in apparently good health, he suddenly had a convulsion, followed by two others, and by complete paralysis of right side with inability to speak; on admission, cannot speak at all; moves leg a little, but arm is motionless and anæsthetic; ordered blisters to back of neck and electricity. May 5.—Has taken strychnine with no benefit; now walks about, but can't speak. June 26.—Uses a very few words; otherwise no change. Discharged by request.

XXXVIII. G. B., æt. 45, admitted July 14th, 1843. Patient has been suffering from rheumatism for some weeks. About ten days ago he suddenly fell down in an apoplectic seizure; has not uttered a word since. His right side is entirely paralyzed. July 22.—Has been getting feebler. Died; no autopsy allowed.

XXXIX. N. G., æt. 39, seaman, admitted April 12th, 1842. Patient has been ill over one year. At that time had an apoplectic stroke, followed by right hemiplegia and loss of speech, from which he has gradually recovered. Is received for obscure disease (cellulitis) of right lower extremity. Had violent hiccough and gradual failure. April 13th.—Died. Autopsy, nine hours after death, showed cerebral substance firm and natural. In left corpus striatum, a cavity the size of a large pea, containing fluid; none in ventricles. Leg was enormously swollen, dark and covered with gangrenous bullæ. No obstruction found in arteries or veins; cellular tissue and muscles easily torn; no heart or kidney lesions.

XL. J. Y., æt. 28, tailor, admitted May 18th, 1843. Six weeks ago had epistaxis, which lasted three weeks, (!) and upon this ceasing while in bed, he had an apoplectic stroke, losing motion and sensation in right half of body; speech completely lost; within two days has spoken a little. June 1.—Is not much improved. Discharged; improper object.

XLI. Mary M., æt. 49, married, admitted January 14th, 1843. Had apoplexy on January 12th. On admission, has paralysis without anæsthesia, on right side; mouth is drawn to left. Seems to comprehend questions, but speaks in a hesitating manner. Discharged February 15th, by request.

XLII. Oliver R., æt. 36, seaman, admitted December 25, 1818, for hemiplegia. A short time before he was suddenly seized with a pain in left side of head, and fell down in a fit. He recovered from this but gradually, having lost the use of the muscles of the right side of face (body meant); mouth and tongue are drawn to left side; right eye has photophobia; articulation is much disturbed. Has equal command over upper and lower extremities. Was purged actively. On 19th of December, while at dinner, he suddenly had vomiting; sweated profusely in evening and complained of cold. At night he became comatose. December 20.—Has been bled without effect; died. Autopsy; great serous effusion under arachnoid. About middle of the anterior lobe of left hemisphere an abscess was found, which extended externally to the dura mater, and appeared internally connected with the lateral ventricle. It was about four inches in circumference, and contained a dirty colored fluid. The surrounding brain substance was much diseased and broken down. A portion of the cerebellum was indurated, the dura mater covering it appearing to have been inflamed. Thoracic and abdominal viscera normal.

XLIII. A.F., æt. 48, painter, admitted September 15th, 1818. One week ago was suddenly affected with loss of sensation and motion in right side of body: "a great impediment in his speech soon followed." On admission is as above; left pupil dilated. September 28th.—Discharged relieved. This patient was seen later, when he had completely recovered.

CASES IN WHICH APHASIA OCCURRED WITH LEFT HEMIPLEGIA.

XLIV. J. J., æt. 40, seaman, admitted June 9, 1850. Has been working in fire-room of steamship at night; on 5th inst., after having headache several days, he went in evening to water-closet, and, while there, had a severe pain in shoulder, and pricking sensation over entire right side; found that he had lost power over left. On admission has complete paralysis, and slight anæsthesia of left side of body, "utterance is imperfect." October 15th.—Has had tonics and electricity; is discharged cured.

XLV. J. B. M., æt. 38, book-keeper, admitted Sept. 25, 1855. On 22d., while going to dinner, he suddenly fell; he did not lose consciousness, and had no convulsions. On trying to rise, he found his left side paralyzed, and was unable to call for assistance; next morning was found and taken care of. On admission, the left side is totally palsied, but not anæsthetic; face turned somewhat to one side; "has some slowness and difficulty of speech." He seems to have enjoyed good health previously, and had been regular in habits; may have had occasional headache. October 18th.—Is discharged, relieved.

XLVI. J. S., æt. 35, soldier of 61st Regiment, N. Y. volunteers, admitted September 20, 1861. Patient was transferred from State Hospital; has been hemiplegic on left side, and has lost speech; is now able to walk about and help himself. No treatment excepting good food. January 20, 1863.—Patient is transferred to United States Army General Hospital in this city.

CASES IN WHICH APHASIA OCCURRED WITH DOUBLE HEMIPLEGIA.

XLVII. J. B., æt. 62, porter, admitted December 6, 1860. Brought in semi-comatose, with left pupil dilated and fixed; face flushed; pulse irregular; body relaxed. December 9.—Patient is rather more conscious; tries to protrude tongue when asked; left side of body is completely paralyzed, right partially so. Ordered wet cups to temple and pot. iod., .60 t.i.d. 12th—Patient now moves right side pretty freely and left slightly. He understands all that is said to him, but cannot speak. Takes beef-tea and pot. iodid. January 7.—After last note patient got bad bed-sores, and last week was attacked with facial erysipelas; in spite of supporting treatment, he died to-day. No autopsy.

XLVIII. R., æt. 41, seaman, admitted September 2d, 1841. About one month ago, while lying in berth, he suddenly became paralyzed on both sides at the same moment. Since, he thinks that he has gained some power over legs. September 14.—Has been taking iod. potass. Last night he was attacked with spasmodic twitching of all his limbs and right side of face.

Cannot swallow as well as before, and his speech has entirely failed. He appears to understand, and attempts to speak. Tongue is protruded; sphincters paralyzed. Cont. iod. potass. September 27.—Has had a good deal of rigidity of limbs; and has gradually failed; died. Autopsy, eight hours post mortem, head only examined. The superficial vessels are congested; some 65. cc. of limpid fluid in cells of pia mater. The arachnoid at base was cloudy and tough. When transverse sections of the brain were made, it was found to be of a natural consistency. The medullary portion was covered with numerous points of blood; nothing peculiar was discovered in hemispheres. The ventricles contained about 15. cc. of serous fluid. The right corpus striatum was found diseased. Its cineritious portion for the space of a hickory nut (2. centimeters in diameter), was transformed into a soft, reddish vascular pulp. The left was healthy, as were theoptic thalami. The rest of the brain was healthy.*

CASES IN WHICH APHASIA OCCURRED WITHOUT HEMIPLEGIA.

XLIX. T. R., æt. 35, seaman, admitted June 14, 1860. Patient has always been healthy; somewhat intemperate; never had syphilis. This afternoon, while working, he fell down unconscious. On admission, is insensible; had no convulsions; pulse 90; no paralysis; heart healthy. June 15.—Has slowly returned to consciousness. There is a complete loss of speech; "muscles of his tongue and of speech are entirely paralyzed, so that he cannot protrude his tongue nor utter a syllable. His intellect appears to be slow, though he evidently comprehends everything." Answers by signs of head. Is able to walk about; eats and sleeps well. June 30.—Continues to improve; is slowly regaining speech, but cannot protrude tongue. July 13.—Patient now protrudes tongue well, and speech is slightly less difficult. Discharged relieved. (If the patient spoke at all, his mutism must have been owing to something besides tongue paralysis.)

L. K., æt. 21, seaman, admitted November 14, 1866. Patient gave an uncertain history of intermittent fever, contracted a month or so before in one of the Gulf ports. On admission, he spoke in a peculiar manner. The first words of any sentence came forth easily and naturally, but the last, often including the important verb or noun, were either gotten out with difficulty, or not at all. The articulation was perfect, but there was a notable failure of memory of which he seemed aware. He had no headache, and no sign of paralysis. He presented the appearance, so commonly seen in patients coming from the South, designated as malarial cachexia. November 15.—Patient seems to have fallen into a curious lethargic state. The speech is almost entirely lost; but there is neither delirium nor coma; he is intelligent, understanding and answering gestures; he makes use of a few words, but never to the point; he seems to wish to be let alone; urine and fæces passed spontaneously, and are normal; the symptoms do not resemble those of congestive malarial fever; the eyes are not remarkably brilliant, and he does not seem to have headache; the pulse ranges about 100; nothing abnormal heard in heart or lungs; is taking .75 of quiniæ sulphas under the skin, daily, in three doses; whiskey 16. cc. and beef-tea. 19.—In the last few days there has been no marked change in his condition; he often makes use of the phrase "what is it?" sometimes says "yes" without reason; still seems rational; puts out his tongue by imitation and quickly too; has been restless, but at no time delirious; two or three times a day he makes use of a few other words; once yesterday saying five in answer to a question, but not forming any sentence; he seems to have rather more fever, with but small morning remissions; bowels being constipated, are to-day moved by injection; when he has a call of nature he demands the nurse's help with the phrase "what is it?" Cont. quiniæ and food; seems to be failing in spite of this; tongue coated, but never dry. 23.—Fever seems to have assumed a more continued type; no change in cerebral symptoms; no paralysis; no delirium; speech same; has had infusion of serpentaria instead of quinia lately. 24.—Rather suddenly, at 4 o'clock A.M., died.

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* These cases are open to the objection that they might have been instances of complete paralysis of the tongue. But in case XLVII., no difficulty in swallowing is mentioned; and in case XLVIII., it is positively stated that the tongue was protruded. Dr. Jackson's opinion on the bearing of such an objection to cases of aphasia is worth reproducing. "I may say, too, that I have never seen paralysis of the tongue (*i.e.* decided paralysis. . . .), even on one side, with pure hemiplegia. There is, generally, weakness, affecting all the four limbs, although the limbs on one side may be weaker than on the other. I state this as a mere matter of fact, but it is easy to explain it byanatomy. The nuclei of the ninth nerves are imbedded very near the decussation of the fibres from the limb, so that an injury involving the nucleus even of one side, would be, from continuity, likely to damage the motor fibres of both sides of the body—those on one side before, and those on the other after they had crossed." And he adds in another place, that dumbness (paralysis of the tongue) would always be accompanied by difficulty in deglutition or by the impossibility of the act.

The autopsy, nine hours after death, was made with great care. On removing the calvarium, the dura mater was found much congested, the arachnoid remarkably dry, and the vessels on the convexity of the brain greatly distended with blood; not a trace of lymph was here found. On examining the base of the brain, however, a large patch of false membrane was found extending from the optic commissure backwards over the crura cerebri, the pons Varolii and the medulla oblongata, terminating in a thin film on the inferior surface of the cerebellum. The membrane was thickest over the pons, being there 2.5 millimeters through and very firm. None of the nerves appeared altered, nor was there any change in the brain substance beneath the membrane. Anterior lobes (particularly the left with the third convolution and island of Reil) were examined with minute care for evidences of softening, inflammation, hemorrhage or plugging of vessels, but nothing could be found. Horizontal section showed considerable punctate injection. The lateral ventricles were about half filled with a clear, straw-colored serum. The whole encephalon was then finely sliced, but no farther lesions were discovered. Lungs, spleen, liver and kidneys were congested. Heart was examined, but condition is not stated in notes.

The results in these fifty cases were as follows: died, 14 (9 autopsies); cured 11; relieved 13; not relieved, 12; total 50.

CASE OF TRICHINOSIS.*

J. B., a grocer's clerk, aged nineteen years, was admitted into the New York Hospital, service of Dr. Thomas F. Cock, June 19, 1867, suffering from anasarca. The patient stated that he had been ill three weeks, and that during the first ten days he had suffered from violent vomiting and diarrhœa, followed by great debility and by dropsy. On admission he appeared extremely anæmic; the œdema was so remarkably elastic that pitting could only be produced upon the legs; the tongue was clean, smooth, devoid of epithelium, and in its anterior part disposed to dryness; the stomach was irritable, he said, but he managed to retain at least one meal a day; during the past few days constipation had succeeded to the previously relaxed state of the bowels; the pulse was weak, small, and beating about 112 times a minute; respiration was normal, and there was no increase of bodily temperature. The urine was at once examined, and found to be of a pale color, of normal specific gravity, and perfectly free from albumen. The patient was ordered 120. cc. of sherry wine, milk diet, and a small dose of the ammonio-citrate of iron in tincture of cinchona. My suspicions that the case was one of trichinosis were strengthened by the negative results of the urinary examination. Direct questioning elicited the fact that considerable muscular pain and soreness had been experienced during the second and third weeks of illness; and that previously to the attack he had been in the habit of eating half-cooked ham in sandwiches, and also of occasionally cutting off small pieces of raw ham in the grocery and eating them.

On the 28th the patient had gained a little strength, vomiting having occurred a few times. Eggs and milk had been eaten, and 180. of wine drunk daily. The tartrate of potassa and iron had been substituted for the citrate, as a chalybeate less likely to disturb the stomach. Repeated examinations of the urine had been made with uniformly negative results. On stating to Dr. Cock my reasons for venturing a diagnosis of trichinosis, he very kindly gave permission to resort to the proving examination. The patient readily and intelligently gave his consent. Localized anæsthesia was produced by means of ether and Richardson's apparatus, and a small portion of the deltoid muscle was removed. On tearing apart the muscular fibres in diluted glycerine and looking at the preparation with a low objective, ten or twelve living trichinæ were seen. The cysts were very distinct, located in the muscular substance, their perfectly limited rounded ends beginning to become opaque from granular deposit. About their extremities were masses of fat globes and cells.

The trichinæ which were still inclosed in cysts presented a constant vermicular motion; while those which were free in the liquid of the preparation were so active as at times to leave the field (a narrow one at one jump. These movements were retained by the trichinæ, thus prepared, for four days.

It was Dr. Cock's opinion, that as all symptoms of intestinal irritation had ceased, and as it might be presumed that the parasites had all reached a muscular lodgment, there were no indications in the case, excepting to feed the patient carefully and to give him iron cautiously. This plan was carried out, and slow improvement was observed daily in color and strength; the swelling disappearing rapidly from all parts excepting the legs. August 13.—The patient, although still pale, is discharged cured.

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* From the *New York Medical Journal*, May, 1868. This was probably the first case of trichinosis in America where the diagnosis was confirmed during life by the microscope. The case occurred while Dr. Seguin was House Physician of the Hospital.—R. W. A.

It is to be regretted that the duties of the hospital were so pressing upon the resident staff as to prevent the proper inquiries and examinations so essential to the completeness of such a case.

A CASE OF ACUTE OCULAR ŒDEMA—CAUSE UNKNOWN.*

B., a private in Co. B, 3d U. S. Cavalry, aged 18 years, a native of Pennsylvania. Has never enjoyed good health. From the age of 15 to date of enlistment, last March, he worked in an iron mill, and was exposed to great heat and vivid light. A few years ago he had intermittent fever. Has always had a tendency to diarrhœa, and is now under treatment for an attack that threatens to become chronic. He is pale, flabby, looks old, face and forehead wrinkled. When a child he suffered from night blindness. Sept. 24th, he presented himself with his *left* eye much swollen; says that ten minutes before his eye was well. He was reading, when suddenly a severe smarting pain was felt in the inner canthus, and was at once followed by swelling. He is certain that no foreign body entered the eye. The lids are externally œdematous and nearly closed. On opening them, an almost complete chemosis is seen, the ring being imperfect at its upper outer part. There is no injection of blood-vessels and no lachrymation; pupil normal. I applied a solution of nitrate of silver (3. to 30. aq.) freely over the chemosis, and directed a light wet compress to be applied during the evening and night. The next day the eye was about normal.

Second attack, Oct. 3d, at 1 P.M. I had occasion to see a wounded man in my hospital, and asked several questions of B., who was watching him. His eyes were then in a normal state. Hardly had I returned to my room—certainly not more than five minutes—than B. came to me hurriedly with his *right* eye swollen. He had experienced the smarting, and the appearance of the eye was precisely similar to that of the *left* eye ten days previously. There was no sign of inflammation. At 2 P.M. a complete and heavy ring of swelling surrounded and nearly buried the cornea. I pursued the same plan of treatment as before, and in twenty-four hours all was well.

Patient states that he had the first attack of this curious affection in 1860, while at work in a field. Since, he has had from two to six attacks each year. This year he was affected once in April and once in July, besides the two attacks detailed above. He feels certain that in all instances the symptoms and appearances have been the same; and often the disease subsided spontaneously. Once, in 1860, both eyes were affected at one time.

His mother and sister "have always had weak eyes," and have suffered

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* From the N. Y. *Medical Record*, Jan. 1, 1869.

from repeated similar attacks of œdema of the eyes. His father has excellent sight, though he has worked for many years in the iron mill.

The irides are pale gray ; pupils habitually a trifle dilated ; urine contains no albumen ; heart healthy. Patient suffers much from epistaxis, in summer mostly.

There are three main points of interest in this case. *First*, the extremely sudden formation of the œdema and absence of the usual symptoms attending inflammatory processes.

Second, the apparent hereditary character.

Third, the coincidence of the disease with a semi-cachectic state.

CAS DE LÉSION PROBABLE DE LA MOITIÉ LATÉRALE DROITE DE LA MOELLE ÉPINIÈRE, DANS LA RÉGION CERVICALE INFÉRIEURE, AYANT PRODUIT DE L'ANESTHÉSIE D'UN COTÉ ET DE LA PARALYSIE DE L'AUTRE.*

Le sujet de cette observation, le capitaine R., du 3^{e} régiment de cavalerie, dans l'armée des États-Unis, âgé de 36 ans, naturellement très-fortement constitué, a toujours joui d'une santé exceptionnellement bonne. Étant en garnison à Natchez, dans l'État du Mississipi, pendant l'automne de 1863, il lui arriva un accident qu'il décrit en ces termes: Il était dans l'habitude chaque jour de se promener à cheval. Le 21 octobre, prenant cet exercice, il aperçut une rue dans laquelle il n'était jamais entré, et s'y lança au grand galop. A peine y fut-il entré qu'il vit que c'était une impasse. M. R. ne put arrêter son cheval, et tous deux furent précipités dans un fossé ayant des murs de briques et d'environ 15 pieds de largeur et 12 de profondeur. Ce fossé terminait l'impasse, et de l'autre côté se trouvait une maison.

Malgré la force terrible du choc, le cheval ne fut nullement blessé, mais M. R. fut relevé insensible et porté à l'hôpital militaire.

Environ une heure après l'accident, il revint à lui et constata sa condition. Il n'avait qu'une seule blessure, une lacération du cuir chevelu sur le pariétal gauche, mais son corps était si complétement paralysé à droite qu'il ne pouvait même remuer un seul doigt. Dans ce même côté la sensibilité était conservée, et il pense même que les sensations tactiles étaient plus distinctes qu'à l'état normal. Dans le côté gauche du corps il y avait anesthésie incomplète, avec conservation du mouvement volontaire. La perte de sensibilité était totale dans les parties animées par le nerf cubital. La tête était fortement inclinée vers l'épaule droite. Il est certain qu'il n'y avait point de paralysie à la face, et que ses sens étaient normaux. Pas de paralysie des sphincters ni d'anaphrodisie. Le peu de dyspnée qu'il ressentit les premiers jours se dissipait lorsque ses épaules étaient soulevées. Il ne perdit plus connaissance et n'eut pas de délire.

Le troisième jour, le mouvement volontaire lui revint un peu dans le côté droit, et après cela il s'améliora progressivement. Vers la fin de la deuxième semaine il quitta son lit, avec l'aide du garde-malade, et fit quelques pas dans la salle en s'appuyant sur le dos d'une chaise ou sur le bras du garde-malade. Une semaine plus tard il se promenait lentement, faisant peu de pas à la fois, tout seul. A cette époque (troisième semaine), il s'aperçut pour la première fois que la sensibilité des doigts de la main gauche s'améliorait, mais il ne remarqua pas l'état de la jambe. En sortant du lit, il fit l'observation que le côté droit du corps se refroidissait plus facilement que le gauche, particularité qui persiste encore aujourd'hui. Six mois après l'accident, il se rendit à son régiment, et avec de l'assistance en montant, put aller à cheval. A cette époque, le cou s'était complétement redressé, et depuis lors il a continué à gagner lentement en force et en agilité du côté droit.

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* Extrait de l'Archives de Physiologie normale et pathologique, mars,1870.

Au moment où cette observation est rédigée (juin, 1869), M. R. est en parfaite santé et soutient très-bien les fatigues de la guerre. A pied il a l'air un peu gêné, mais cela tient à un degré très-minime d'atrophie du côté droit du corps. Une fois à cheval, il est complétement dans son élément et se montre admirable cavalier. Sa force physique est grande, et il fait l'exercice du sabre également bien des deux mains ; la main droite est un peu congestionée, et les doigts sont en apparence contracturés, mais cela est plutôt une attitude vicieuse qu'une rétraction musculaire. Avant son accident, il écrivait de la main droite ; maintenant il conserve l'habitude prise pendant les mois de paralysie d'écrire avec la gauche, mais il signe toujours avec la droite. La jambe et la cuisse gauche n'ont pas une sensibilité tout à fait normale, autant que je l'ai pu estimer avec une épingle. Il pense que la main droite est encore hypéresthétique, et il a remarqué que de ce côté les ongles poussaient plus rapidement qu'à gauche.

L'histoire du traitement est plutôt curieuse qu'importante. Les chirurgiens à l'hôpital militaire de Natchez crurent à une lésion dans l'hémisphère cérébral gauche. L'erreur de diagnostic s'explique en partie par la présence des deux faits que voici : plaie sur le pariétal gauche et hémiplégie à droite. Ils décidèrent de laisser M. R. sans traitement, sauf qu'après les premiers jours le galvanisme (courant électro-magnétique interrompu) fut appliqué journellement aux membres paralysés pendant quatre mois. Dans le mois d'octobre 1864, profitant d'un congé, il se mit entre les mains d'un horloger de Louisville, qui lui fit des applications irritatrices produisant des pustules. Il se servait d'un appareil composé d'une douzaine de pointes d'aiguilles, avec lequel il faisait pénétrer une pommade dans la peau du côté droit, qui était encore faible. Toutes les parties de ce côté du corps furent l'une après l'autre couvertes de pustules, le traitement durant plusieurs semaines. M. R. croit que cette médication lui fit du bien, et depuis il ne s'est plus soigné.

Cette observation est malheureusement très-incomplète. Elle repose presque entièrement sur ce que le malade a lui-même recueilli. Je n'ai pu étudier d'une manière satisfaisante l'état des diverses espèces de sensibilité dans le côté gauche, car les détails du cas me furent communiqués pendant une expédition contre les Indiens dans le territoire du Nouveau-Mexique, et ce n'est pas en pareilles circonstances qu'un médecin se trouve muni d'æsthésiomètre, etc.

La première question qui se présente en cherchant à analyser le cas est quelle partie du système nerveux central a été lésée.

On peut complétement rejeter l'opinion des chirurgiens qui traitèrent

M. B. en premier lieu. A moins de supposer deux lésions simultanées, une dans l'hémisphère gauche et l'autre dans la moelle épinière, les symptômes ne peuvent être expliqués. Mais l'absence de paralysie faciale s'oppose à l'admission de l'existence d'une lésion cérébrale. Le malade a souffert des symptômes qui suivent :

A droite.	*A gauche.*
1. — Paralysie complète du mouvement volontaire dans les deux extrémités.	1. — Conservation du mouvement volontaire.
2. — Une contracture (?) de muscles inclinant la tête vers l'épaule.	2. — Une paralysie (?) des muscles dont l'action est d'incliner la tête vers l'épaule.
3. — Une hypéresthésie dans ces mêmes extrémités (?). Certainement conservation de sensibilité.	3. — Anesthésie complète dans la région animée par le nerf cubital, incomplète ailleurs.
4. — Congestion de la peau et développement des ongles plus rapidement que du côté opposé.	

On ne peut s'empêcher de reconnaître ici les signes d'une hémiplégie spinale ; une forme de paralysie dont on doit la caractérisation à M. Brown-Séquard.*

Se basant sur une physiologie de la moelle épinière qu'il avait lui-même, par de remarquables découvertes, plus contribué que personne à établir, il parvint à expliquer les résultats, en apparence si obscurs et indéchiffrables, de certaines lésions spinales. M. Brown-Séquard a récemment † publié une série de leçons sur l'hémiplégie spinale, remplie de faits nouveaux qui ne laissent plus de doutes sur le sujet.

En comparant les symptômes présentés par le capitaine R. avec ceux des malades étudiés par M. Brown-Séquard, il est certain qu'ils appartiennent à une même catégorie. La lésion dans l'observation que je rapporte était donc dans la moitié lateral droite de la moelle épinière.

A quelle hauteur siègeait la lésion? Il est certain que les nerfs diaphragmatiques n'ont point été paralysés, car le peu de dyspnée dont il s'est plaint se dissipait immédiatement après l'élévation des épaules. La sensibilité était le plus affectée dans les parties où se distribue le nerf cubital, nerf qui semble prendre son origine des deux cordons inférieurs du plexus brachial, cordons qui sont formés par la dernière racine cervicale et la première dorsale. Conséquemment la moelle n'était point lésée au niveau de la quatrième paire cervicale, mais l'état pathologique était à son maximum à l'origine de la huitième paire.

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* Lectures on the physiology and pathology of the nervous centers : delivered before the Royal College of Surgeons of London in May, 1858.—Philadelphia, 1860.

† *Lancet*, Nov. 7, 1868, et dans plusieurs numéros suivants.

Quelle était la nature de la lésion ?

Le résultat heureux obtenu si rapidement suffit pour faire rejeter la plupart des causes connues de l'hémiplégie spinale. En premier lieu, une fracture du rachis ne peut être admise ; et de plus, j'ai constaté qu'il n'existait aucune déformation de la colonne vertebrale. Une hémorrhagie des méninges, assez considérable pour produire une anesthésie si persistante, aurait nécessairement été très-considérable, et de plus le sang aurait eu de la tendance à se répandre autour de la moelle, produisant ainsi des symptômes bien différents de ceux observés dans l'observation. Une déchirure complète de la moitié latérale droite de la moelle n'est guère plus compatible avec les symptômes et avec le retour du mouvement volontaire pendant la première semaine. En outre, comme M. Brown-Séquard l'a fait remarquer, l'anesthésie est permanente lorsqu'il exist pareille lésion. Dans un cas qu'il a pu observer plus de vingt ans après la division d'une moitié latérale de la moelle dans la région cervicale supérieure, il a constaté un retour assez complet du mouvement volontaire dans les membres du côté de la lésion, mais à peine y avait-il trace de sensibilité dans le côté opposé. La conduction motrice s'était rétablie, la conduction sensitive était interrompue d'une manière permanente. Il me semble qu'une lésion très-limitée dans la substance grise de la moelle, au niveau de la huitième paire cervicale, lésion consistant en une dissociation de certaines cellules d'avec leurs fibres nerveuses, avec rupture de vaisseaux capillaires et hémorrhagie extrêmement minime, expliquerait parfaitement les symptômes. Mais, en outre, il faut faire la part d'une altération de nutrition dans d'autres parties de la moelle, altération conséquente au traumatisme. La paralysie, qui a disparu complétement en quelques mois, ne pouvait dépendre entirèment d'une interruption des conducteurs moteurs ; il a dû y avoir aussi une altération de nutrition par action réflexe. La sensibilité est revenue en presque totalité, aussi avec une grande rapidité ; et d'après les preuves fournies par M. Brown-Séquard de l'incurabilité de l'anesthésie à la suite de lésions de la substance grise de la moelle, on doit conclure que l'anesthésie chez M. R. dépendait presque entirèrement de l'altération de nutrition dans la moitié droite de la moelle. Mais il y a certainement eu traumatisme, ainsi que l'atteste la persistance d'un degré appréciable (peut-être plus grand qu'il ne m'a paru) d'anesthésie. L'état congestionné de la main droite, ainsi que la production anormale des ongles, indiquent une altération permanente dans la substance grise.

Comment expliquer la position de la tête pendant plusieurs mois ? Deux hypothèses s'offrent ; une de supposer une paralysie des muscles du cou, du côté gauche ; l'autre, de considérer la déviation comme due à un spasme des muscles à droite. La première hypothèse ne peut être soutenue, il me semble, parce qu'une paralysie des muscles latéraux du cou aurait presque certainement entraîné une diminution dans le mouvement de l'épaule et du bras du côté correspondant ; et M. R. dit positivement qu'une heure après l'accident il avait l'usage complet du bras gauche. La contracture des muscles à droite pourrait s'expliquer par l'altération de nutrition dans la moelle au-dessus de la lésion. On ne pourrait la considérer comme un épiphénomène, dû à la violence du choc. Il me semble que la première opinion est plus en accord avec les connaissances que, grâce aux travaux de M. Brown-Séquard principalement, nous avons de la production de phénomènes multiples et variés par une seule cause d'irritation.

AUTOPSY OF A CASE OF MANIA.*

Mania; granular degeneration of ventricles; cortical sclerosis of cord.—S. S., æt. 43, married, Can., stonecutter, admitted July 10, 1870. First symptoms noticed two months ago. Had a slight fit, which was followed by symptoms of subacute mania. He steadily grew worse and became unmanageable at home, on account of violence towards family and others. At times has been rational, but most of the time incoherent, having delusions in regard to being a policeman, owning livery stables, etc. Previously to his having this "fit" he was thought eccentric.

August 9.—Has had no active symptoms until to-day, when he became delirious and required restraining in bed.

Sept. 1.—Is up and about ward as usual, though he seems more stupid and irritable.

20th.—Transferred to the "Excited Ward" on account of a growing disposition to molest other patients. Imagines that they call him bad names, etc. Patient is somewhat deaf.

Oct. 10.—General health failing. Is taking tonics and stimulants. Soils clothing and bed.

Nov. 1.—Did not seem to recognize his wife, who visited him two or three days ago. Frequently suffers from colic.

11*th.*—At six o'clock A.M., was discovered in a "fit" by the attendant. The "fit" seems to be apoplectic. Has stertorous breathing; flushed countenance; pulse 132, and very strong, carotid arteries beating violently; pupils contracted and not responsive to light. No reflex movements of limbs produced by tickling feet. When first seen by attendant arms were jerking. No evident paralysis of any of the limbs. At 6 o'clock P.M., no material change. Has occasional spasmodic movements of arms, and spells of jerking, and irregular breathing. Sweats profusely, and passes large quantities of urine.

12*th.* A.M.—Pulse not as strong. The paroxysms of irregular respiration continued through the night. No other change. Noon.—Breathing more regular; pulse better; pupils respond to light; he makes attempts to swallow. Ordered some stimulants. Is sensible to pain of pricking and pinching. Right side apparently more sensitive than left. Temperature normal. 6 o'clock P.M.—Swallows whiskey and water, though with difficulty. Pulse 100.

13*th.*—Apparently better. Takes milk-punch and beef-tea with less difficulty in swallowing. Respiration more regular. 5 P.M.—Is about as in A.M. 7 P.M.—Reported in a dying condition. Death took place at 9 P.M.

Autopsy.—Body examined twelve hours after death; weather cool. Rigor mortis moderate and universal. Some hypostatic congestion of skin of back and limbs. Small bed-sore formed over sacrum.

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* By A. M. Shew, M.D., and E. C. Seguin, M.D. Extract from the American Journal of the Medical Sciences for July, 1871.

Spine and contents.—Fat next to dura mater injected. But little fluid under arachnoid. Dura mater and arachnoid appear perfectly healthy: there being a few filamentary adhesions between the two in region of cervical enlargement, posteriorly. On the anterior surface of spinal canal there is found a small tumor, the size of a large pea, situated in the substance of the posterior common ligament, opposite the fibro-cartilage lying between the last dorsal and first lumbar vertebræ. It pushes the dura mater somewhat forward, but not enough to have produced any pressure on cord. The tumor is fibro-cartilaginous. On making sections through different parts of the cord, no abnormal appearance is discernible to the naked eye, except a very unusual development of vessels in the gray matter and near the anterior fissure. Spinal cord is placed in Müller's fluid for transportation.

Skull and contents.—Bones and dura mater normal. Pacchionian bodies unusually numerous. The arachnoid is not perfectly transparent along vessels, but the milkiness is hardly pathological. A minute inspection of the entire external surface of the cerebrum fails to reveal any lesions. The pia mater everywhere peels off well. On section, the gray matter of the convolutions appears unusually dark. The centrum ovale majus is much injected. Opto-striate bodies normal.

The ventricles are the seat of a very interesting lesion. In the first place, they are all considerably dilated, and contain a good deal of clear fluid.

Secondly.—The floor of the lateral ventricles, half way up the lateral boundary, appears granular and thickened. The granulations, easily felt and seen, are translucent, pearl-like, and vary much in size, from a mere point to the bulk of the head of a small pin. On passing the fingers over the diseased tissue it appears unusually firm. The third ventricle and infundibulum are considerably enlarged, and studded with granulations; the same being true of the aqueduct of Silvius. The foramina of Monro measured over one-half a centimeter in diameter, and the lesion involves them, as well as the septum lucidum. The roof of the ventricles is normal; and so are the choroid plexuses.

Cerebellum appears perfectly normal: but the valve of Vieussens is the seat of the granular degeneration above described. Medulla oblongata appears healthy to naked eye, except that floor of fourth ventricle to the apex of the calamus scriptorius exhibits the granular degeneration, though to a less degree than the lateral and third ventricle. Nerves at base of brain appear normal.

Thorax and contents.—Heart normal, contracted; commencing atheroma of arch of aorta.

The lungs do not fully collapse on exposure. Left lung presents some recent adhesions of its lower part with costal pleura; no fluid. The lower lobe is hepatized and presents a number of patches of commencing resolution; pieces sink in water. Lower part of upper lobe slightly congested; at apex there is a wrinkled cicatrix the size of an almond, underneath which lies a cretaceous mass as large as a pea, with tubercular (?) granulations, somewhat smaller, imbedded in the lung tissue around it, in a zone one inch in diameter.

Right lung exhibits only extreme congestion and œdema of upper and lower lobes; the middle lobe being normal. No trace of tubercles in this lung.

Abdomen and contents.—The stomach is the seat of considerable ecchymosis along the lesser curvature, and there are traces of this in the large cul-de-sac. Kidneys of usual size; capsules peeling off normally. Cortical portion of right kidney is, perhaps, a little pale.

Liver, spleen, and intestines healthy. Some urine in bladder; is not albuminous.

Microscopical examination. Spinal cord.—After hardening in a dilute solution of chromic acid, sections were cut horizontally from different regions of the spinal cord, and prepared by Clarke's method. That is to say, the cuts were stained by a neutral solution of carmine, soaked in alcohol, transferred to absolute alcohol, floated on oil of turpentine to give transparency, then mounted in Canada balsam dissolved in chloroform. The following is the result of the study of these sections in order, beginning above, with a power of one hundred diameters.

Section No. 1, made in the decussation of the pyramids. Circumferential layer of reticulum is perhaps a trifle heavier than usual, as are some of the trabeculæ running inward from it. The central canal is very small, but not altered; and no lesion is evident in the white or gray substances.

Section No. 2, made 3 c. below decussation. The increased thickness of the circumferential part of reticulum has become very decided, and constitutes a cortical sclerosis. The framework is denser, and secondarily the nerve fibres near the edge of the section are atrophied in large numbers; many bearing but a very small quantity of myeline around the axis cylinder, many others being apparently reduced to the latter element; the growth of the new fibrillar tissue having, as it were, strangulated them. The anterior fissure is widely open, the vessels running into it are abnormally large, and at its bottom, near the anterior commissure, there is a moderate amount of homogeneous effusion in the folds of the pia mater outside the vessels. On the left side, the anterior commissure is invaded, and somewhat broken down, by a dilated vessel with effusion of the same homogeneous material outside of it, in a manner precisely similar, though less in extent, to what exists in Section 4. The central canal is normal, but its cavity contains a quantity of the homogeneous effusion.

Section No. 3, made through the upper part of the cervical enlargement. The cortical sclerosis has diminished, but is still very evident and uniform. Near the bottom of the anterior fissure lies a dilated blood-vessel, and still deeper a quantity of homogeneous effusion, destroying, in great part, the left anterior commissure. There is also a want of symmetry between the anterior horns, the right being shorter, and looking a trifle more inward, than the left. The central canal is small, but normal.

Section No. 4, made through the middle of the cervical enlargement. The cortical sclerosis has again increased, and is greatest in posterior and lateral portions of the section. At the external end of the anterior fissure, there has been much increase of the connective tissue of the pia mater. With a higher power (300 diameters), this structure may be seen sprinkled with numerous nuclei, evidences of inflammatory irritation. This multiplication is especially marked round about the vessels of the anterior fissure, and in their adventitious coat. A narrow strip of pia mater extends from this part to the inner end, or bottom, of the anterior fissure, where it again expands, rich in nuclear elements, and bearing abnormally large blood-vessels, whose walls are decidedly thickened, and whose cavities are crammed with red blood corpuscles. In the middle, resting against the anterior commissure, in the midst of the connective tissue, is a mass of effused material, appearing homogeneous under a power of 100 diameters. From the pia mater, at the bottom of the fissure, there extends a new formation of fibrillar tissue filled with nuclei, inclosing blood-vessels, which has quite destroyed the left branch of the anterior commissure; the pathological product extending into the gray matter and posterior commissure. At the place of contact of the fibrillar tissue and the gray commissure, is another mass of homogeneous effusion. There is more or less condensation of tissue round about the central canal, the cavity of which contains a homogeneous material of same aspect as that found elsewhere. The invasion of the left anterior commissure by the diseased pia mater has produced a striking deformity in the white and gray matters of that side. The left anterior column is shortened, and rounded off below; the inner boundary line of the anterior horn is apparently shortened, and is thrown inward, so as to appear quite parallel with the anterior fissure. About one-fourth of the so-called posterior fissure is dilated, and the resulting space contains a thickened process of the pia mater, probably itself conveying an enlarged blood-vessel.

Section No. 5, made through the middle of the dorsal region. The cortical sclerosis continues marked, though less than in No. 4. At the bottom of the anterior fissure is some homogeneous effusion, but the commissure is intact. There is seen, back and to the left of the central canal, in the posterior commissure, an opaque red spot, contrasting sharply with the adjacent tissue, of an oval shape, measuring .21 by .13 mm., evidently a spot of sclerosis. The central canal is here very large, .5 mm. by .15 mm., irregularly quadrangular in outline, and is filled with homogeneous effusion.

Section No. 6, made through the middle of the lumbar enlargement. Cortical sclerosis moderate; exaggerated near points of exit of posterior roots. The anterior fissure and contained parts are very nearly normal; anterior commissure intact. The central canal appears as a slit running antero-posteriorly, one of its sides formed by tolerably distinct epithelium, the other broken down; and the lumen contains *débris* of epithelial elements. No trace of effusion outside of vessels.

Section No. 7, made at a point about 2 c. above end of cord. Shows very great cortical sclerosis, which is, however, quite uniform. Anterior fissure not much involved; it contains no effused matter. The central canal is represented by an ovoid mass of epithelial *débris*. The external layer of the reticulum, and the trabeculæ running inward from it, are immensely hypertrophied, and show, under a higher power, a distinct fibrillar structure. The nerve-fibres are consequently atrophied, and very few exhibit an envelope of myeline; the immense majority are indicated only by the round, nucleus-like body, the axis cylinder. Besides these, there are very numerous nuclei, belonging to the diseased reticulum. Beyond the external layer of reticulum are seen two fragments of pia mater, much heavier than normal, and showing nuclei.

Section No. 9, made at a point 1 c. above end of cord. The cortical sclerosis is here very considerable, the pia mater being remarkably thickened. The sclerosis is especially marked at the external end of the anterior fissure. In the fissure itself there is thickening of the pia mater, enlargement and thickening of blood-vessels; and around these there is some effusion more granular than that occurring higher up, and of a yellowish hue. Opposite the posterior fissure the sclerosis is much less intense; but it is again greater over the lateral columns. Around the central canal there is some slight condensation of tissue, the epithelium is fairly preserved, and there is no effusion in the cavity. The effusion which has been seen in so many sections, lying in the anterior fissure or occupying the lumen of the central canal, appears quite homogeneous, and of a uniform reddish hue, under a power of 100 diameters. With a power of 340 diameters, we thought that in a few places we could make out indistinctly the outlines of red blood corpuscles. We consequently, though with reservation, consider the effusion as hemorrhagic.

In none of the many sections examined was any alteration of the nerve cells of the gray matter discovered. In some sections the gray matter was seen to contain abnormally large vessels filled with blood.

The state of the spinal cord may be stated in a few words, as follows:

The organ is in a state of inflammatory irritation, characterized by thickening of the pia mater, multiplication of its nuclei, and formation of new fibrillar tissue in the anterior fissure in various parts of the cord, mainly in the upper cervical region and in the lower portion of the lumbar enlargement. This morbid state of the pia mater is accompanied everywhere by enlargement of blood vessels and by thickening of their external coat; in many places, besides, by rupture of small vessels, leading to effusion of blood in the bottom of the anterior fissure throughout the upper cervical region. In some places the central canal is occupied by a similar effusion. In the upper cervical region for several c. the left arm of the anterior commissure is broken up by a pathological product made up of enlarged vessels, new fibrillated tissue derived from the pia mater, and by hemorrhagic effusion. Throughout the cord there is marked cortical sclerosis. This sclerosis, most marked in the upper and lower portions of the organ, has resulted, firstly, in the production of a mass of fibrillated tissue containing numerous nuclei, and, secondly, in consecutive atrophy of the nerve fibres lying in the meshes of the reticulum near the periphery. In the gray matter, traces of the irritative process are to be found in the shape of condensation of the tissue round about the central canal, of enlarged blood vessels, and of one nodule of sclerosed reticulum.

Medulla Oblongata. We have been as yet unable to study the state of this part in a thorough manner. Sections made at a point .5 c. above the end of the calamus scriptorius present the following points:

The pia mater is somewhat thickened, and contains numerous nuclei. The floor of the ventricle is studded with granulations, most abundant and largest in the situation of posterior median fissure, having the same fibrillated structures as those found on the lateral and other ventricles, a minute description of which will be found below.

The nuclei of the hypoglossal nerves, and the cells of the restiform bodies, are normal ; but a little to the outside of and above the hypoglossal nuclei, there are seen six or eight nerve-cells in various stages of granular degeneration. Some of them are mere masses of yellow granular pigment. These cells belong to the nuclei of the spinal accessory nerves. The vessels of many parts of this nervous centre are very large, and nearly all crammed with blood corpuscles. In and about the hypoglossal nuclei, vessels measuring .15, .1, .06, 0.5 mm. in diameter are found ; in the restiform bodies, vessels 0.5 mm. in diameter ; and in one section the central part of the left olive exhibits a cavity which must have inclosed a vessel measuring .3 mm.

Ventricular Surface.—We come now to the study of the most interesting lesion of the case, viz., the granular degeneration of the general ventricular surface. We shall describe the lesion as it appeared in sections cut from the floor of the lateral ventricle perpendicularly to surface, prepared and mounted according to Clarke's method.

(*a.*) On viewing such a section with various powers, the following points are ascertained : The deeper parts of the section exhibit the nuclei of neuroglia in normal numbers, and the ordinary number of blood-vessels, around which there is a moderate deposit of yellow granular matter, this being the only abnormal appearance. Next, immediately underlying the epithelium, is a layer of condensed tissue, contrasting quite sharply with that above described, and measuring, on the average, nearly .5 mm. in thickness. At several points, in the neighborhood of blood-vessels, this condensed tissue penetrates deeper into the normal brain substance. These vessels, in the condensed layer, and in the parts immediately below, are abnormally large, filled with blood corpuscles, and their coats are evidently thickened. The upper free (epithelial) edge of the section is quite covered with projections, these being sections of the granulations whose appearance to the naked eye has already been described. These sections vary much in outline and in size. Some present a distinct terminal nodule or head, others are cut off squarely, a few are pointed, and many are rounded. They range in height from a mere nothing to nearly .2 mm. The majority of the granules appear opaque, and the opacity extends somewhat, and in various shapes, into the subjacent tissue. In some places nothing is visible but a nodule of darker appearance than the surrounding parts, and not actually projecting through the limiting line of the section.

(*b.*) On viewing one of the non-projecting nodules with an objective magnifying 340 diameters, it is evident at the first glance that the epithelial lining of the ependyma remains over the entire nodule, in a better or worse state of preservation ; in some places the nuclei of the epithelial cells can be distinctly seen. In reality there are here two nodules lying very near each other, a larger and a smaller one, and in the depression between the two a certain amount of disintegration has occurred in the epithelial layer. The nodules themselves appear made up of a confused mass of delicate fibrillar tissue ; the parts underlying the nodules being made up of similar fibres nearly horizontally disposed. The nodules appear separated from the other tissue by a tolerably sharp outline ; and no cellular elements are visible beneath the epithelium.

(*c.*) Examining one of the large projecting granulations, we see that there is, as above described, a fibrillated substratum, horizontally disposed. The epithelium is preserved on the limiting line or section on either side of the granulation, and also for a short distance upon the granulation itself. The granulation, made up of fibrillated tissue, the fibrils of which are disposed perpendicularly to the edge of the section, projects in such way through the remains of epithelium as at once to suggest that the growth has burst through the once continuous layer of epithelial elements. Its free edge is made up of delicate fibres, and among these are seen the outlines of oval nuclei. In the deeper parts of the granulation the fibrillation is more confused, and the nuclei no longer distinct.

(*d.*) The examination of a minute granulation at its free edge shows a number of peculiarly shaped cells, containing very large granular nuclei, and having dissimilar ends; one narrow and thread-like running down into the granulation, the other free, rounded, or squared off. A number of analogous cells appear faintly outlined in the projecting part of the granulation below its free edge. These peculiar bodies are most probably altered epithelial cells of the ciliated variety.

(*e.*) The head or projecting part of a granulation being snipped off with scissors, is teased to pieces in dilute chromic acid solution ($\frac{1}{1200}$), and placed under an immersion objective of 1.25 mm. focus made by William Wales of Fort Lee, N. J. It is at once perceived that what, under a power of 340 diameters and excellent definition, appeared as a fibrillar connective tissue, is resolved under 1,000 diameters into a congeries of minute nerve fibres, the majority of which bear a small quantity of myeline, the others being apparently naked axis cylinders, or amyelinic fibres, measuring on the average .001 mm. Nothing whatever in the field resembles fibrillar connective or reticulated tissue. There are present lying among the fibres a small number of cells and nuclei. Some of the nuclei are free, round, presenting a sharply defined outline, measuring from .005 to .007 mm. in diameter, and inclosing a prominent granule or nucleolus. The cells are ovoid, without membrane, somewhat granular (not pigmented), and exhibit nuclei similar in size and appearance to those described above. These cells measure in their long diameter, on the average, .014 mm.; in their short diameter .01 mm. In addition to these elements, there are three very peculiar, and we must admit puzzling, cells. These bear a resemblance to modified epithelial cells of the type described at *d*, but the long and slender extremities branch in a regular and remarkable manner. The heads of these cells present an irregular ovoid outline, measure in length about .018 mm., and transversely (short diameter) .01 mm. One of them contains a nucleus precisely similar to the nuclei above described. The prolongations or tails of the cells present no demarcation line from the heads, and taper gradually, giving off two to six branches. From the further rounded end of a cell to the second subdivision of its tail, is a distance of .07 mm. Some of the smallest branches of these cells, by their uniform diameter, .001 to .0015 mm., sharp outline, and homogeneous appearance, bear the most striking resemblance to amyelinic nerve fibres.

From this study we think it safe to say that in this patient the ventricular lesion was not due, as in Mr. Lockhart Clarke's case, to proliferation of the epithelial layer of the ependyma. The preparations all show a very distinct sub-epithelial lesion, whether of a truly sclerotic nature we cannot positively state. In favor of sclerosis we have the increase of density in the granulations themselves and in the underlying tissue to a depth of .5 mm., with thickening of the coats of blood-vessels and enlargement of perivascular spaces, in the same part. Against this view there may be advanced the absence of modified reticulum or neuroglia, which is said * to constitute so large a proportion of the ependyma ventriculorum. Had we contented ourselves with using a power of 340 diameters, we should have honestly asserted the existence of fibrillar tissue, as making up the bulk of the granulations. The uncertainty of our study of this lesion has demonstrated to us the great want of a new investigation, with modern objectives, into the normal histology of the walls of the ventricular cavity.

Sections made through the aqueduct of Silvius exhibited granulations of precisely similar constitution. It will be remembered that in the preparations from the medulla oblongata, granulations of the same appearance were seen to spring from the floor of the fourth ventricle.

The lesion of the general ventricular cavity may be summed up as follows: A condensation of the sub-epithelial tissues, with perivasculitis and dilatation of the vascular canals, to a considerable depth. A similar condensation immediately under the epithelial cells, affecting the form of nodules, which nodules in course of growth have burst through the epithelium, and projected into the ventricular cavity. In their development these nodules have, furthermore, set up an irritation in the adjoining structures which has resulted in a modification (proliferation ?) of the epithelium itself; this last being, we firmly believe, a secondary and subordinate process.

Cerebellum.—Nothing abnormal is discernible in sections of the convolutions of this organ.

Cerebrum.—Sections cut from one of the convolutions of the right anterior lobe, and from one of the inferior part of the right temporo-sphenoidal lobe, show no lesion beyond the presence of a few yellowish granulations along the blood-vessels.

We will only add a few words of an historical nature concerning the granular degeneration of the ventricular walls.

First observed by Bayle,† this lesion does not seem to have been observed with care until 1861, when Dr. J. Lockhart Clarke,‡ in studying the alterations present in a case of progressive muscular atrophy, found the fourth ventricle studded with granulations, of which he gives the following concise account:

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* Virchow, Cellular Pathology, pp. 311–14 (Am. ed.).

† Bayle, Traité des Maladies de Cerveau et de ses Membranes. Paris, 1826, p. 464. (Quoted by Clarke.)

‡ Dr. J. L. Clarke and Dr. Gairdner, Relation of a Case of Muscular Atrophy, Beale's Archives of Medicine, vol. iii. p. 1, 1861. London.

> "The whole floor of the fourth ventricle, as already remarked, presented a very peculiar and unnatural aspect. Instead of being smooth and shiny, as in the healthy state, it was entirely paved with a multitude of granulations or small rounded eminences, which were very closely aggregated, but differed from each other considerably in size. I removed some of them for examination, first by scraping them off from the surface, to which they adhered with some tenacity; and then by shaving off a section together with a thin layer of the subjacent tissue. When examined by means of a sufficiently high magnifying power, the granulations or eminences were seen to consist of globular aggregations of the ordinary epithelial cells, which in a natural or healthy state, are arranged side by side, and form a smooth or level surface on the floor of the ventricle. The tissue immediately subjacent, and which consists of exceedingly fine fibres proceeding from the tapering ends of the epithelial cells and running in various directions, was more abundant than usual; and—as might be expected from the homologous relation of this part to that which surrounds the spinal canal—they were interspersed with corpora amylacea, but certainly not to a corresponding extent."

The same granular condition of the ventricular surface was, about the same time, attracting the attention of a French alienist, M. Joire, who, early in 1861, submitted to the Paris Academy of Medicine* a paper in which he stated that he had found this condition only in cases of general paralysis of the insane, and advanced the view that this was a characteristic lesion of the disease. In the *Gazette Médicale* for 1864, page 528, is an abstract of a second paper by M. Joire, published in the *Bulletin Médicale du Nord*, in which he describes the appearances to the naked eye of these granulations. He states that the parts underneath the epithelial layer are softer and more translucent than usual. This condition often coincides with dropsy of the ventricles, and subarachnoid effusion. In early stages the granulations are small, numerous, and remind one of grains of sand. In old cases the granulations are larger, whitish, or transparent, and produce a feeling of roughness under the finger. The lesion is most common over and round about the calamus scriptorius. Finally, M. Joire claims that the lesion is constant in general paresis. In this abstract there is no evidence whatever of the microscope having been used.

Griesinger,† in his classical treatise, merely observes that in chronic hydrocephalus the ependyma of the ventricles is very often found covered with granulations, thickened and denser than normal, and as resisting as leather.

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* Joire. Bulletin de l'Académie Impériale de Médecine, séance du 19 Février, 1861, p. 395.

† Griesinger. Traité des Maladies Mentales (traduction du Dr. Doumic). Paris, 1865, p. 496.

We find the following in Leidesdorf:* In senile hydrocephalus there is found a thickening of the ependyma of the ventricles, as part of the general thickening of the neuroglia, giving rise to a granular appearance. Under the head of new formations this author mentions, without details, granulations of the ventricles which are derived from connective tissue.

Maudsley† merely quotes Clarke and Joire, denying the latter's assertion concerning the meaning of the lesion. In Dr. Blandford's‡ new book the following passage occurs:

> "Granulations of the lining membrane of the ventricle have been thought by M. Joire to be peculiar to general paralysis, which they are not. They have been observed in old-standing cases of mania or dementia, together with similar granulations of the pia mater of the parietal and occipital lobes and medulla oblongata. They are, no doubt, an aggregated and abnormal condition of the epithelial cells, and seem to contain a homogeneous substance, probably exuded lymph."

Rindfleisch § speaks of granulations, like dew-drops, occurring on the ependyma of the ventricles, more particularly upon that of the fourth ventricle, in cases of chronic hydrocephalus, epilepsy, masticatory spasm, disorders of speech. He considers them as made up wholly of fibrillar connective tissue, and a very few cellular elements.

Finally, we have been informed by Dr. Francis Delafield, one of the curators to Bellevue Hospital, that during the last two years he has met with this lesion about a dozen times, nearly always in connection with granulations in the pia mater. He has not, however, made any microscopical examination of these products.

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* Leidesdorf, Lehrbuch der psychischen Krankheiten. Erlangen, 1865, s. 256.

† Maudsley, The Physiology and Pathology of Mind. Second edition. London, 1868, pp. 455–6.

‡ Blandford, Insanity and its Treatment. Am. ed., Phila., 1871, p. 121.

§ Rindfleisch, Lehrbuch der Pathologischen Gewebelehre. Leipzig, 1867–69, s. 546–7.

CONTRIBUTIONS TO THE PATHOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.[*]

I. *Examination of the Cervical Sympathetic Nerve in a Case of Unilateral Sweating of the Head.*—Some attention has of late years been paid to this interesting symptom, and various explanations of its mode of production have been offered.[†] In many cases mechanical interference (pressure of tumor or aneurism) with the sympathetic nerve is readily made out. In others, the patients' antecedents and the results of treatment warrant the diagnosis of a sympathetic process, one associated with disorder of the stomach or other distant organ. But there are still other cases, in which, the above explanation failing, we are obliged to have recourse to the unmeaning phrase of idiopathic disorder. The instance which I reproduce was of this last kind, and to my knowledge is the only one of its class in which a microscopical examination of the sympathetic chain has been make.

The subject observed by me (a male about 50 years of age) had exhibited one-sided sweating of the face and neck for a considerable number of years; in which period the right half of the face and neck never showed any moisture, not even when the left side was bathed in perspiration. The abnormality, therefore, consisted in the abolition of sweating on one side of the head. When the patient first came under my observation he was in a cachetic state, which ultimately proved to be cancerous, an abdominal tumor soon becoming apparent. On the evening when the patient's wife first informed me of the one-sided sweating, he had been suffering severe abdominal pain, and, in consequence, was perspiring nearly everywhere. The lower and upper limbs and the trunk were moderately moist, the left side of the face (forehead especially) was dripping wet; while immediately beyond the median line the skin of the right side was perfectly dry. The pupils were equal in size and in mobility. A careful estimate of the surface temperature on the temporal region of either side was made by means of Dr. E. Seguin's surface thermometer, but gave no differential result.

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[*] From the Am. Journ. of Med. Sciences for October, 1872.

[†] Consult a good paper on the subject by Roberts Bartholow, M.D., in the *Quarterly Journal of Psychological Medicine*, vol. iii.(1869) pp. 134-144.

On the 7th of May the patient died of exhaustion. The autopsy was made on the 8th, sixteen hours after death, the body having been perfectly preserved in ice. Immense cancerous masses were found in the abdominal cavity, affecting principally the mesenteric glands. Another deposit, in the shape of a rounded tumor as large as a small orange, was met with behind the left clavicle, externally to the sterno-mastoid muscle. Microscopic examination showed the tumor to be composed of carcinomatous tissue.

The sympathetic chain of the neck was carefully removed on both sides, and very shortly afterward immersed in an artificially iodized serum. During the dissection it was observed that the right nerve was unusually adherent to the sheath of the vessels and pneumogastric nerve, from a point on a level with the bifurcation of the carotid artery nearly up to the superior sympathetic ganglion. The post-clavicular tumor did not involve the left nerve in any way. Examination May 9th, twenty hours after the autopsy. To the naked eye the right chain exhibits no middle ganglion, and presents a marked injection of the nerve just above the superior ganglion, in part corresponding to the adhesions already described. The left chain appears absolutely normal (has three ganglia).

Microscopical examination of a considerable number of preparations from the right superior ganglion, teased in serum, or stained with carmine: no granular or amyloid bodies can be detected, nor any abnormality of the connective tissue; the nerve fibres are normal, and the nerve cells alone depart from the healthy standard. This alteration consists in a marked increase of the granular yellow pigment, which normally occupies one-sixth to one-fourth of the body of the ganglion cells. In many cells the pigment is more than equal to one-half the cell bulk; in quite a number it takes up nearly the whole cell body, and almost conceals the nucleus. Preparations from that part of the nerve which should have presented a ganglion contain abundant ganglion cells, nearly all with an abnormal amount of pigment. Only a portion of the inferior ganglion remains; but preparations from that show precisely similar appearances. Similarly prepared specimens from various parts of the nerve trunk exhibit perfectly normal nerve fibres, nearly all myelinic, of very variable diameters. No evidence of proliferation of the connective tissue can be seen. The various vessels met with in the above preparations contain large quantities of blood-globules, but their coats are healthy.

Left nerve.—Nearly every part of this chain is examined in the same manner; its nerve fibres, blood-vessels (not injected), connective tissue, and ganglion cells appear in precisely the same state as similar elements of the right chain, *i.e.*, the first three are perfectly normal, the last are very granular.

Consequently, the only lesion found in these nerves is symmetrically developed, and can, consequently, bear no relation to the one-sided arrest of sweating observed during life. In another case it would be highly desirable to examine the cervical portion of the spinal cord as well as the sympathetic itself.

The following measurements are obtained from estimating the size of very numerous elements on both sides. Ganglion cells range from .025 mm. to .062 mm. in diameter; nerve fibres from .001 mm. to .02 mm.

II. *Double Central Canal in part, of an otherwise normal, Spinal Cord.**—While examining sections from various parts of the spinal cord of a patient who had died of the affection known as general paresis of the insane, I came across unmistakable evidences of the existence of two central canals, most distinct in the cervical enlargement.

(*a.*) In a section made just below the apex of the fourth ventricle, the central canal is single and open, exhibiting very distinct cells. Its long diameter, antero-posterior, equals .53 mm.; its short diameter .052 mm.

(*b.*) In a section from a point 3 c. below decussation of pyramids, a single central canal, blocked up by distorted epithelial cells, is seen.

(*c.*) Section from the upper part of the cervical enlargement shows two small, beautiful central canals, lined with nearly perfect epithelium. The interval between the two canals equals .03 mm. The posterior median fissure comes down to a point opposite the middle of this interval. The lumen of one canal measures transversely .049 mm.; antero-posteriorly .018 mm. The transverse diameter of the lumen of the other canal measures .07 mm.; the antero-posterior .012. The epithelial cells have an average length of about .01 mm. The epithelium is entirely preserved around the left canal, but is a little frayed at the inner angle of the right. Between the canals is a shapeless mass of cellular bodies, such as usually lie about the central canal.

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* Instances of Double Central Canal. G. Harley and Lockhart Clarke in a case of acute myelitis. *Lancet*, October 3d, 1868, p. 451.

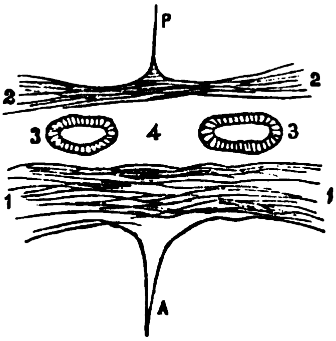

Diagram of Median Part of Transverse Section of Cervical Cord. P. Posterior median fissure. A. Anterior median fissure. 1. Anterior commissure. 2. Posterior commissure. 3. Central canals lined with distinct epithelial cells. 4. Interval filled up with dense cellular structure such as surrounds central canal usually.

(*d.*) A section from the lower part of the cervical enlargement shows traces of two canals, but the epithelium is not in as good condition, and a lumen is distinct only on one side. Intervening and surrounding cellular structure not as dense as in (*c.*)

(*e.*) Section from the mid-dorsal region exhibits a confused mass of cells in the center of the commissure, with one distinct, irregular aperture, and another very imperfect one at a distance of .08 mm.

(*f.*) In the lumbar enlargement nothing is found but one common cellular (not epithelial) central mass without lumen.

(*g.*) At a point 3 c. above end of cord no distinct epithelium or canal is visible.

CASE OF GENERAL PARESIS OF THE INSANE.*

Alexander C ——, aged 40 years, born in New York, a machinist by occupation, married. Admitted Jan. 7, 1870.

History.—His wife states, that about five years ago he had an attack of tonsillitis, after which, for a short time, he manifested symptoms of exalted mania. Until the date of the illness now reported, his health was, as usual, good, but he was more irritable and impatient than formerly. He had been employed as skilled workman by the Weed Sewing Machine Company, at Hartford. In August (1869) engaged himself to the Singer Co., in New York. This change proved the starting point of a series of extravagant notions and excesses, which manifested themselves during the following month. Previous to this unusual conduct his wife had no idea that he was not perfectly sane. She recollects, however, that for some months before leaving Hartford he had manifested, at times, an unusual degree of sexual desire, that he ate enormously, complaining afterward that he had not had enough food.

The principal exaltation symptom, which developed in New York, consisted in imaginary business connection with James Fisk, Jr., in railroad matters. He went to the Opera House to look for him, and once got up and stopped the orchestra. Thought that he was heir to A. T. Stewart, and to Astor, whose daughter he was to marry. He became intemperate and visited houses of ill-fame; purchased expensive clothing, jewelry, etc., and nearly ran through his means in a few weeks.

On admission to the hospital he had extravagant notions of wealth and of power; was to erect hotels in every city of the world, and to grant any wish to anybody and make everybody happy.

May 29th.—Has once or twice shown violence toward the patients.

Examination, June 25th.—I copy from my notes: Comes into the room in a quick way, unconscious of our presence, exclaiming that he is the greatest potentate on earth, the father of Jesus Christ, etc. Is perfectly docile and obedient, but every motion is hurried and jerky; after answering a question he constantly and immediately reverts to his exalted ideas. General muscular development very good. Heart sounds normal; percussion and ausculation of anterior and upper part of lungs give natural results; no urinary disorder.

Head.—Hearing on both sides, fair; has slight convergent strabismus dependent on weakness of right external rectus muscle; pupils, normal in size and motion; has no nystagmus. Vision of left eye good, but denies reading time on full-sized watch at a greater distance than 40. cent. with right eye. [On July 24th an examination with the ophthalmoscope, by Dr. T. R. Pooley, of New York, gives the following results: "Outline of both optic discs irregular; discs themselves small and white; vessels, especially arteries, small in size. Atrophy subsequent to optic neuritis."] No facial deviation, and none of tongue when projected. Frequent fibrillar contractions are visible in orbicularis palpebrarum, in orbicularis oris, and in levator anguli oris. Speech markedly thick, especially so after declaiming—labial sounds affected. Projects tongue fairly, and though the organ does vibrate as a whole, there are no fibrillar contractions visible in it, and no wrinkling.

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* Report of the Pathologist of the Connecticut Hospital for the Insane, Dr. E. C. Seguin, for the year 1872.

Upper Extremities.—Slight trembling of hands and fingers when extended, a vibration being perceived by observer when holding latter slightly between his own hands. No fibrillar contractions are visible in arms, and none can be excited by filliping. Strength very great and about equal in both hands; the efforts being made in a peculiar, jerky manner. The dynamometer (of Tiemann's make) is too weak to record strength. In the letters which he occasionally writes, the handwriting appears irregular, but to-day, with care, he signs his name in quite a free, firm hand for a man of his station. Sensibility is normal in its various modes, save that there is a little slowness (sensorial or mental?) in his perception of heat. Co-ordinates well in executing every movement; eyes open or closed. Measurements of extended arms—circumferences, right (middle) 26 c., left 24.3 c.; forearms flexed at right angles, measured near bend of elbow, right 27 c., left 26.5 c.

Lower Extremities.—Walks well, but in a jerky manner,* produced by mental state. Stands and walks securely with eyes open or closed. Right leg at thickest part, extended, measures 35.5 c.; left, 34.5 c. Sensibility in its various modes, normal, save that there is a retardation in perception of pain and heat. Muscles of entire body exhibit normal electro-muscular contractability and sensibility.

The nurse states that C. never soils his bed or clothing, and that he does not masturbate.† His delirium runs exclusively upon greatness and power of self.

Diagnosis.—Chronic diffused peri-encephalitis, or general paresis of the insane, in tolerably early stage.

July 20th.—Has been rather more excited during the past ten days; is full of delusions of an exalted character; articulation more impaired.

August 2d.—Is, and for several days has been, violent, almost unmanageable. Temperature observations begun. Agitation of hands again investigated. On holding C.'s extended fingers lightly between our own extended hands, a peculiar parchment-like fremitus is communicated to us, apparently a result of friction between the contiguous sides of patient's fingers, brought about by fibrillar muscular contractions. On comparing this condition with that present in a well-marked case of tremulous hands and head (paralysis agitans?) the following are results obtained: In the case of tremulousness, the movements consist in alternate flexion and extension of fingers, due to contractions of whole muscles, or of large bundles of fibres; on holding this patient's fingers in the manner above described, no fremitus is perceived, and the movements are wholly controlled.

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* This expression refers to action of body as a whole in the act of walking; there was no jerky movement of legs.

† Another attendant says that patient has masturbated freely.

February 19th, 1871.—Since last note disease has progressed. Delusion and delirium of most exalted sort have continued. Has gradually emaciated. No epileptic seizures. Two months ago passed into a well-marked, peculiar adynamic state, in which he now lies. A number of decubitus sores have developed quite rapidly on several projecting parts. Emaciation is extreme, but uniform; perhaps most marked in face. Eyes in same condition as before, right with slight strabismus. Pupils of about normal size (compared with those of a healthy person in the same light), right a trifle larger than left; both responding to light. Movements of orbicularis oris slow, stiff, but complete. Projects tongue pretty well, without deviation; organ does not appear wrinkled, and trembles but little as a whole; presents no fibrillar contractions. Articulation very imperfect; he can be understood rarely, and with difficulty. Swallowing of liquids fairly performed; a little cough coming on after six or eight spoonfuls have been taken. Nurse reports greater trouble than this at times, indicated by "choking spells." Movements performed by all limbs slowly and feebly. *No trace of inco-ordination* or trembling; no fibrillar contractions anywhere visible. Sensibility cannot be satisfactorily studied. Patient makes no complaint of bed-sores, and did not call attention to two or three abscesses which formed some time ago. Pinching is everywhere felt and complained of, though after some seconds of delay. Heart's action exceedingly feeble, though regular; pulse, 100 per minute. Respiration not counted, performed fairly, without râles. Urine and fæces passed involuntarily, but not unconsciously. Mind is as clear as compatible with presence of peculiar exalted delusions, which are still well marked—"is the first man born," "has boundless wealth," etc. About the middle of the night C. died rather suddenly.

Autopsy.—Nine hours post mortem (Feb. 20th). Temperature of room in which body has lain has been below 10.° C. Extreme uniform emaciation. Rigor mortis, moderate. Ulcerations on under aspect of both elbows, on sacrum, and on spines of scapulæ. An abscess is discharging on internal face of left foot; one, unopened, exists in inner side of left thigh. No syphilitic stains.

Skull and Contents.—Bones healthy. No abnormal adhesions of dura mater. Considerable opaline sub-arachnoid on convexity of brain, compensatory of atrophy of anterior lobes. Veins of convexity abnormally full. A large quantity of fluid lies at base of brain, and when body is laid prone a fresh gush takes place from the vertebral canal. Olfactory ganglia unusually firm and gray. Optic nerves appear normal; so do the third nerves. Membranes at base of brain normal. Fifth and other cranial nerves normal. Spinal membranes and external aspects of cord and medulla oblongata normal. Vertebral and basilar arteries are not atheromatous, and contain recent dark clots.

The floor of the fourth ventricle appears healthy. Cerebellum healthy. Cerebrum: Pachionian bodies moderately developed. Marked atrophy of convolutions composing anterior lobes—greatest in front. A small amount of fluid exists in the lateral ventricles; their floors are healthy. Opto-striate bodies normal. Various sections through spinal cord show no abnormal appearance.

Thorax and Contents.—The fifth rib of right side presents an old united fracture. Between the sixth, seventh and eighth ribs, on left side, are a succession of abscesses, burrowing in each intercostal space. Opposite the sixth space the peritoneum is firmly adherent. The fibres of the diaphragm are healthy.

Lungs.—Slight adhesion of entire lower lobe of left lung to parietal pleura; right lung free, except over diaphragm. Some fresh adhesions between all lobes of right lung. A few patches of lobular pneumonia exist in apex of right lung. The only other lesion is a slight congestion of the left lower lobe.

Heart.—Opaque patch with granulated surface on parietal pericardium. Yellow appearance of muscular substance in range of left coronary artery. On the outer surface of each ventricle is a small white patch. The aortic valves are healthy. A firm clot exists in aorta, the beginning of which exhibits patches of atheroma. Firm clots lie in right auricle; and a large white and black one in left auricle.

Abdomen and Contents.—Liver pale, slightly bronzed on under surface, and presents a number of yellow patches. Gall bladder nearly empty, containing a little bile. Liver substance perhaps a little fragile. Pancreas healthy. Mesenteric glands unusually large. Descending colon, as it lies from the lower edge of kidney to the brim of pelvis, is contracted to the size of a man's ring finger. Transition from normal to contracted portions, sudden. Caput coli and appendix vermiformis normal. No obstruction of intestines. Left kidney: its cortical portion is broad and whitish; lymph in pelvis. Right kidney weighs 135. grams; same as left, but whiter. Suprarenal capsules are unusually large, 63 mm. long by 31 mm. wide, but not diseased. Bladder vascular. Body of left biceps contains a rounded tumor. No evidence of periostitis anywhere.

Eyes removed and placed in Müller's fluid. The sympathetic chain in the neck, on either side, is removed, and preparations made in artificial serum, and with carmine. These examined with suitable powers of the microscope show none but normal nerve cells and fibres, and no increase in the connective tissue uniting these elements is noted. Preparations from the sympathetic in the left side of chest, from the greater splanchnic nerve, and from the semilunar plexus give similar results.

Examination of the Cerebrum after being four days in Müller's Fluid.—Secondary gyri numerous. Left hemisphere: the convolutions of external surface of anterior lobe are narrower than normal, the sulci widened; these changes being most marked in the anterior part of the inferior and middle gyri.* The superior frontal gyrus is well preserved, as is the posterior part of the inferior, bordering upon the fissure of Silvius. Orbital lobule much dwarfed anteriorly. Distance from anterior edge of corpus callosum to extremity of anterior lobe is only 33 mm. Nothing worthy of note on surface of rest of hemisphere, except that the superior gyrus of temporo-sphenoidal lobe (that bordering on fissure of Silvius—posterior marginal of Broca) is atrophied, and that the parietal lobe shows rather wide sulci. On section, the gray matter of affected gyri appears more shallow than that of the healthy convolutions. No gross lesions are discoverable in vessels or brain substance. On the right hemisphere the same general appearances are exhibited, except that the inferior frontal gyrus is rather better preserved. Superior gyrus of temporo-sphenoidal lobe very small. In parietal lobe, about the termination (upper end) of the intraparietal sulcus, anteriorly to postero-parietal lobule, there exists a remarkable interval between the gyri, quite unlike anything figured as normal by Gratiolet. The gray matter of convolutions of anterior lobes is as thin as in other half of cerebrum. The apex of anterior lobe is even more shortened, the distance from anterior border of the corpus callosum to extremity of lobe being but 32 mm. No coarse lesions of nerve tissue or vessels can be seen.

Microscopical Examination of the Nervous Centres.—After seven days' immersion in Müller's fluid, the state of the elements making up the cerebrum is investigated by means of teazed preparations stained in carmine, put up in glycerine or in dilute chromic acid (one part acid to fifteen hundred parts water). (*a*) Preparation of gray matter of a convolution of the left occipital lobe shows nerve cells with nuclei, and processes quite normal; small vessels and many capillaries filled with blood globules, their walls healthy; no granular bodies or pigment masses. (*b*) Preparation from left anterior lobe, second convolution above orbital gyrus in great longitudinal fissure, exhibits normal (slightly granular) cells and blood-vessels. (*c*) A specimen from cortex of apex of left temporo-sphenoidal lobe shows normal nerve cells; and some few blood-vessels whose walls contain scattered granules. (*d*) In a preparation from the first convolution anterior to fissure of Rolando, near longitudinal fissure, left hemisphere, vessels are found crammed with red blood corpuscles; their coats are healthy, and there are no granular bodies. Right hemisphere, preparations from (*a*) convolution of occipital lobe in great longitudinal fissure, (*b*) from anterior lobe in longitudinal fissure, (*c*) from in front of fissure of Rolando, near longitudinal fissure, (*d*) from apex of temporo-sphenoidal lobe, show nothing abnormal except the presence of a few (scattered) granules in the walls of many vessels, including capillaries. Preparations from the middle of the corpora striata exhibit abundant yellow granular deposit in coats of blood-vessels, in the adventitia, and in the perivascular space; the nuclei of many capillaries are granular. Many vessels are filled with blood.

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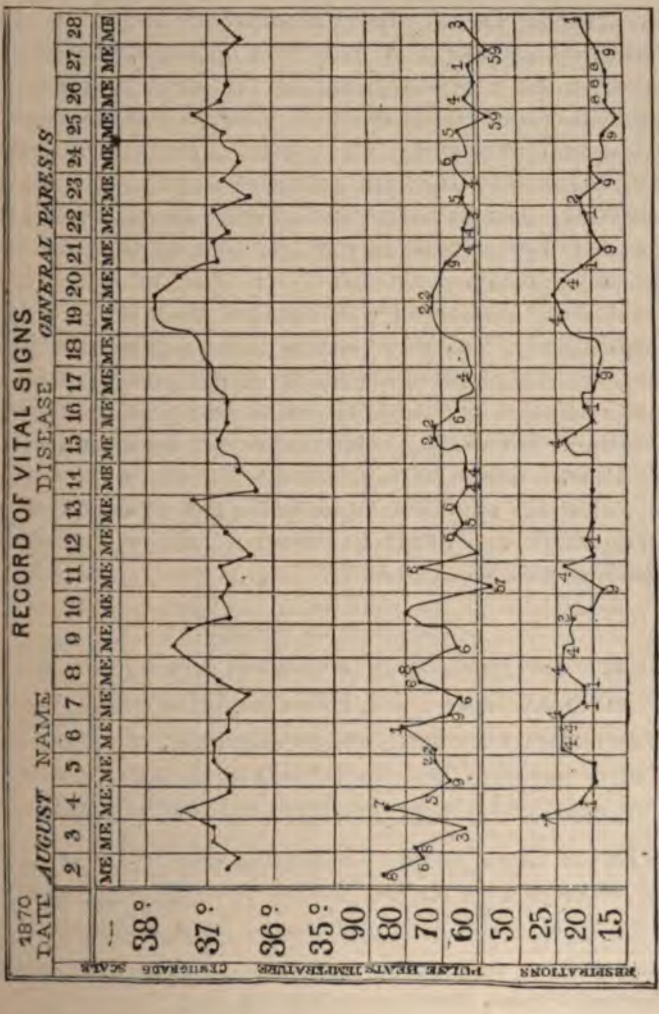
* In this account the classification of convolutions of Prof. Turner, of Edinburgh, is followed.

Examination of the Hardened Nervous Centres.—The spinal cord was cut into pieces about two centimeters in length, and suspended in a chromic acid solution (one part to five hundred) for about six weeks, when it was transferred to strong alcohol. Sections were subsequently cut from these pieces, stained with carmine, soaked in alcohol and in absolute alcohol, cleared up in oil of cloves, and mounted in a solution of Canada balsam in chloroform. Many sections from each of the points specified below are carefully studied with low and high powers of the microscope. (*a*) Sections from a point 3 c. below decussation of pyramids, (*b*) from the upper part of the cervical enlargement, (*c*) from the lower cervical region, (*d*) from the mid-dorsal region, (*e*) from the lower part of the lumbar enlargement, and (*f*) from a point 3 c. above termination of cord. These preparations appear normal in every particular. No trace of granular bodies can be found (posterior columns being searched with especial care), the nerve cells, the nerve fibres, and neuroglia are everywhere normal. The medulla oblongata is examined in sections made at the following points: (*a*) at the apex of the fourth ventricle, (*b*) through the middle of the olivary bodies, (*c*) 5 mm. below lower edge of pons Varolii, (*d*) 3 mm. below edge of pons, through striæ of auditory nerve, (*e*) 2 mm. below edge of pons, (*f*) through lower part of pons, showing nucleus of sixth and seventh nerves. These sections present nothing whatever abnormal, except a slight dilatation of some of the largest perivascular canals. The brain was left in a mixture of bichromate of potassa and an uncertain quantity of a solution of chromic acid (one part to one hundred). After several weeks the pieces of the different lobes of the brain were transferred to strong alcohol. [I may here state that in cutting the brain to prepare these pieces, no gross lesions were found in any central part.] Sections were cut from the hardened pieces, including the pia mater wherever possible, and treated in the same way as the cord sections. Many of these preparations are attentively studied, with the following meagre results: (*a*) Preparations from the convexity of right hemisphere, near the termination of fissure of Rolando, at the great longitudinal fissure. Pia, torn off; nerve cells, normal; many perivascular spaces enlarged, but containing no pathological products; vessels themselves, and neuroglia, apparently unchanged; no granular or amyloid bodies. (*b*) Preparation from the right anterior lobe; pia evidently much thickened, with production of much fibrillar connective tissue and many nuclei; in the pia meshes there may be seen scattered yellowish pigment masses, which bear no definite relation to the blood-vessels; the nuclear new formation is especially abundant about the vessels. The gray matter exhibits vessels which have lymphatic sheaths rather narrower than usual, and within the sheaths lie a few pigment granules. Nerve cells and neuroglia appear normal. White substance contains capillaries and larger vessels gorged with blood corpuscles; none of the capillaries showing any degeneration of their nuclei. No recognizable changes in neuroglia and nerve fibres. (*c*) Preparations from the orbital convolution of the right anterior lobe, exhibit essentially same lesions as above. In addition, some vessels of the cortex are tortuous in their sheaths. (*d*) Sections from the left anterior lobe (different part) present alterations similar in kind and extent. (*e*) Preparations from the apex of occipital lobe appear absolutely normal. (*f*) Sections from the convolutions of the cerebellum present a normal appearance. (*g*) Sections made perpendicularly to the floor of the lateral ventricles show epithelium and underlying layer absolutely normal. (*h*) Transverse sections of third nerve (motor oculi) are normal. Dr. Delafield reports that the optic nerve is absolutely normal, and that the eyeball presents no alterations worthy of note.

The following measurements of the patient's temperature, pulse, and respiration were made by Dr. W. B. Hallock, as near as possible to the hours of 9 A.M. and 5 P.M.



INFANTILE SPINAL PARALYSIS.*

GENTLEMEN:—I think that you may obtain a clearer idea of the importance of infantile spinal paralysis if we study it not separately, as I had at first purposed we should, but as one of the forms of paralysis to which infants are liable. At a subsequent meeting we can briefly inquire into the other varieties of infantile palsy, and you will then obtain a knowledge of a great part of the nervous pathology of infancy, and be better prepared to diagnose the most important of these affections—infantile spinal paralysis. The period of life termed infancy is that which includes the first dentition to the beginning of the seventh year. The pathology of this stage of existence is treated of separately by authors, in part because of the peculiar difficulties in diagnosis presented by children of this age, and in part because of numerous peculiarities they exhibit, such as susceptibility to the action of causes of disease, of medicines, and the exaggeration of certain groups of symptoms, such as the spasmodic element in the sphere of the nervous system. There is no essential difference, please remember, between infants and adults in the nature, course, and treatment of disease.

The following synopsis represents the principal forms of paralysis which are likely to occur in infancy, together with their leading pathogenetic factors:

PARALYSIS IN INFANCY.

1. Hemiplegia (clot, injury, embolism, tumor in brain).
2. Paraplegia (injury, clot, inflammation of cord).
3. Peripheral paralysis (compression, etc., of nerve trunk).
4. Inhibitory paralysis (peripheral irritation).
5. Infantile spinal paralysis (atrophy of motor nerve cells).

The last of these is the disease we shall study to-day, and several examples of it are before you, affecting subjects of different ages. The term which I have adopted is one of numerous synonyms by which the disease has been known. The first systematic writer upon the subject, Heine, used a corresponding German name, *spinale Kinderlähmung*, in his second edition of 1860. French writers upon the diseases of infancy, who studied the subject very thoroughly in its clinical aspects, Rilliet and Barthez,* denominated the disease *paralysie essentielle de l'enfance.* Bouchut, entertaining a very singular notion of the pathology of the affection, gives it the name of *paralysie miogénique.* Niemeyer retains the term essential paralysis, considering it as a safe one to adopt during the present unsettled state of opinion upon the question of pathology. The word *essential* is used by these authors as equivalent to functional, or *sine materia.* The title infantile paralysis is one employed by such authors as C. B. Radcliffe and Adams. Duchenne, in the last edition of his work on electrization, calls the disease *paralysie atrophique de l'enfance*, instead of *paralysie atrophique graisseuse de l'enfance*, as in his edition of 1860. Hammond, in his treatise upon Diseases of the Nervous System, speaks of organic infantile paralysis.

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* From the Medical Record, N. Y. 1874. A lecture delivered at the College of Physicians and Surgeons, New York, on Nov. 8th, 1873.

SYMPTOMS AND COURSE OF THE DISEASE.

In brief infantile spinal paralysis may be defined as an acute febrile affection, resulting in generalized paralysis, which shortly disappears from all but a limited part of the body, where the akinesis persists indefinitely without impairment of sensibility, is accompanied within a few weeks by atrophy of the palsied muscles, and is followed later by various deformities—the result of altered balance of power at certain joints. The anatomical lesion of the disease consists in primary (?) atrophy of the nerve cells of the anterior horns of the spinal cord (motor tract), and in secondary (?), complicating (?) myelitis.

There are three quite distinct stages in the course of infantile spinal paralysis.

(*a*) *The Febrile Stage.*—A child, and usually a healthy or even a robust child, has an indisposition, with febrile movement, evidenced by restlessness or drowsiness, hot skin and frequent pulse. The mother's attention is often called to a co-existing local affection, such as indigestion, intestinal catarrh, or morbid dentition. The last is the usual condition referred to, because the age when infantile spinal paralysis is most apt to occur (six to twenty-four months) includes nearly the whole period of dentition, and because mothers as well as many practitioners are only too willing to hold the teeth responsible for any disease which may arise during their evolution. The fever has not, to my knowledge, been studied with the thermometer. Fever has been observed in nearly all cases* (forty out of fifty). When parents assert that no fever was present at the outset, it must be borne in mind that a certain degree of intelligence is required to enable a person to recognize fever; that in some cases only skilled observations (thermometrical measurement) will reveal its existence; and that it may have been present for a few hours only. Besides these phenomena, there are several nervous symptoms present in a certain proportion of cases, convulsions, usually not involving the facial muscles, and delirium. Hyperæsthesia is also present; but whether this is a true hyperæsthesia, or an evidence of that morbid sensitiveness which is part of the febrile state, remains uncertain.

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* Rilliet et Barthez, Maladies des Enfants, t. ii., p. 545 et seq. Paris.

(*b*) *Stage of Paralysis.*—As the fever subsides, the mother, upon handling the child, discovers that voluntary motion has been lost in nearly the entire body. The exact period when the akinesis appears, and whether it develops slowly or suddenly, are points not yet investigated. The paralysis is at first generalized, often involving all the limbs, very rarely the face. At the same time no impairment of sensibility is to be noted. In a short time, a few hours, the paralysis recedes from some parts and persists in others; it being very rare that more than one half of the body remains palsied. This retrocession of the paralysis, its disappearance from some limbs, and fixation upon one or two, or upon unsymmetrical distant muscles, is highly characteristic of the disease—I might almost say pathognomonic. This fixation is usually upon parts of the lower limbs, more seldom upon the upper, and rarely upon parts of the trunk. In only one case has a muscle of the head been found definitely paralyzed—the temporal. The muscular group which is most often affected is the anterior tibial and peroneal. Owing to the immunity of the abdominal muscles, the bladder and rectum perform their functions.

(*c*) *Third Stage.*—Inseparably connected with the definitive akinesis is the muscular atrophy, with loss of electro-muscular contractility, and with deformities. The loss of irritability in palsied muscles, as tested by the electrical current (faradic), occurs very early—at a period varying from one to six weeks. In one case atrophy and loss of electro-muscular contractility were well marked on the fourth day (Laborde*). A very few years ago three observers independently made the very valuable observation that these atrophied muscles could be made to contract by the application of the galvanic current. Mr. Harry Lobb, † Dr. William A. Hammond, ‡ and Mr. J. Netten Radcliffe § (from 1863 to 1865), are each entitled to the merit of making this observation in infantile palsy. The application of this reaction to the galvanic current to prognosis will be stated further on.

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* Laborde. De la paralysie (dite essentielle) de l'enfance. Paris, 1864, p. 3.

The atrophy of the muscles is both relative and positive, and is accompanied by certain histological changes, which will be detailed when we study the morbid anatomy of the disease. In the case now before you, a young man seventeen years old, in whom infantile palsy set in at the age of twenty months, the bones of the legs, themselves much reduced in size, seem covered only by skin and a little adipose tissue. These legs also exhibit two accompaniments of the atrophy of infantile spinal paralysis, viz., imperfect circulation and diminished temperature. The atrophied parts are very purple, and are quite cold to the touch. The diminution of temperature has been measured by Heine,‖ and found very great; the atrophied region in one case having a surface temperature of only 17° C. This is in striking contrast to the temperature of limbs palsied by a cerebral lesion.

In consequence of this akinesis, indefinitely prolonged, accompanied by wasting of the muscular tissue, there are formed a number of *deformities*. Some of these consist merely in arrested development, as indicated by the shortening of the affected limb; there occurs a relative atrophy. The shortening of a limb, a lower limb more especially, gives rise to secondary deviations in forms, such as twisting of the pelvis and lateral curvature of the spine. These secondary deviations, which are the result of attempts to restore disturbed equilibrium, are called compensatory. It is important to distinguish these compensatory deformities from the primary, or truly paralytic ones, because the treatment of either kind differs radically. The positive deformities are caused, as stated in the definition, by impairment in the balance of muscular power about any articulation. For example, in the feet of one of the patients before you, you see an example of deformity which constitutes the talipes equino-varus of surgeons; toes point downward and inward. The anterior and posterior muscles of the legs are palsied and atrophied, and consequently the feet obey the laws of gravity, dropping into their position because the ankle joint is well back upon the posterior third of the feet. There is no resistance to passive movement in all directions. In other cases only one set of tibial muscles is paralyzed, and the deformities are produced by the unchecked (unantagonized) action of the healthy muscles. In the feet we may thus have talipes equinus ("the most common and important variety," Erichsen), or talipes equino-varus, or any of the varieties which you will find fully described in surgical text-books. In a case of talipes equinus of this sort we find great resistance to any attempt at overcoming the deformity; the tendo Achillis is very tense. The muscles governing the movements of the knee joints may be paralyzed, and in consequence we obtain analogous deviations at that joint. About the upper extremities the deformities are less striking, but are produced by the same double mechanism, obedience of parts to gravity, and unchecked action of certain muscles. The spinal deformities are nearly always of the compensatory sort: for instance, a shortened leg necessitates the inclination of the pelvis to the side on which the shortening is; and in order to preserve the equilibrium of the body, the lumbar vertebræ bend so as to form a concavity on the opposite side, and higher up there occurs a secondary spinal curve to the side of the shortened limb. The lateral curvature is known technically as scoliosis. This is rarely due to palsy of spinal muscles, though I have now under treatment a case of this primary paralytic scoliosis. A variety of muscular disorders may cause scoliosis in individuals not affected with infantile paralysis. (Consult the various works on deformities.) Other spinal deformities, more often primary, are the bending forward of the spinal column, non-angular, called kyphosis; and the bending backward, termed lordosis. The bending forward is shown in a minor degree (quasi-pathological) in what is termed stooping—a supposed sign of studious habits. Lordosis, as first clearly pointed out by Duchenne, may be due to palsy of the abdominal muscles, or to bilateral palsy of the lower spinal extensors; in both cases the upper part of the body is thrown unnaturally backward to preserve equilibrium. The two forms may be recognized without a minute examination of the seat of palsy, by using a plumb-line. Dropping the line from the shoulders, it will clear the sacrum in cases of lordosis due to palsy of the erectors of the spine; whereas in palsy of the abdominal muscles it will fall within the sacrum. In the latter case there is a deepening in the normal lumbar curve without positive backward projection of the upper part of the body.*

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* Laborde. Op. cit., p. 19.

† In a letter to London Med. Times and Gazette, 1863, Vol. ii., p. 682.

‡ New York Medical Journal, Dec., 1865, Vol. ii., p. 168.

§ Ref. by C. B. Radcliffe, in Reynolds's Syst. of Medicine, Vol. ii., art. on Infantile Paralysis, p. 665. London, 1868.

‖ Heine. Spinale Kinderlähmung. Stuttgart, 1860, p. 16.

One word more concerning the genesis of primary deformities. It is often said that in club-foot and other deformities, the efficient cause of the deviation is spasm of the preponderating muscles. This I believe to be extremely rare, and a belief in this erroneous doctrine leads to insufficient (merely orthopædic) treatment of the deformities.

Changes in nutrition in the paralyzed parts; many of these, as observed in the disease, consist in what may be termed relative atrophy, *i.e.*, arrested development or retarded growth, *e.g.*, diminution in the calibre of the blood-vessels, not only of the parts affected, but extending further into the vessels above the atrophied muscles, and even into the main trunks. In a certain number † of cases of atrophied lower extremities, a marked diminution in calibre has been found to exist in the iliacs, and even in the lower portion of the aorta. In the boy before you, with extreme atrophy of all the muscles below both knees, the whole vascular system of the lower extremities is abnormally small. It is necessary to bear in mind that no pathological change is discoverable in the walls of these vessels; it is an excellent example of simple arrested development. Again, we find the bones of the implicated regions diminished in size, the arrested development affecting rather their circumferential than their longitudinal growth, only a slight shortening being found to exist even in cases of long standing. Let us now devote a few moments to the study of the more positive changes in nutrition, as exhibited in the muscles. The morbid anatomy of the muscular system in infantile spinal paralysis has been the subject of laborious and careful investigation, and the results of the latest researches tend to convince us of the existence of different kinds of muscular degeneration, though by no means of equally frequent occurrence, in this disease. By far the most common form is: (*a*) the simple atrophy, a form which almost always affects such muscles as are kept in forced repose for a long period; it manifests itself in a shrinking of the individual muscular fibres; a transverse section reveals the separation of the contractile substance proper from the sarcolemma; moreover, as a rule, we find a fatty infiltration of the cells of the connective tissue. (*b*) A granular degeneration is described as succeeding in order of frequency. In this form proteinaceous granules (bodies unaffected by ether, dissolved by acetic acid) are found in the fibre itself; the striation disappears gradually, more slowly than in the simple atrophy; as in the latter form, a fatty infiltration of the interstitial tissue also exists. (*c*) A true fatty degeneration is believed to be comparatively rare. The earlier belief, that it constituted the peculiar muscular lesion in infantile paralysis, aside from its occasional occurrence, may have arisen from the almost unexceptional co-existence of the interstitial fatty changes with the forms above described.

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* Duchenne. De l'Electrisation localisée. 3me ed. Paris, 1872, p. 498.

† Case by Charcot and Joffroy. Archives de Phys. normale et pathol., 1870. Tome 3, p. 134.

Pathological Anatomy.—We have now to consider the morbid anatomy of the disease itself. By the earlier writers it was believed to be essentially functional in its character. The first step toward giving us a clearer conception of the true nature of the affection was made by Heine, who published the results of his observations in 1840; discarding the functional theory, he expressed his belief that the disease was due to a violent congestion, with perhaps a subsequent inflammation of the nervous centres. Later, cases were published by Laborde and Cornil, in which a sclerosis of the antero-lateral columns was described. In an autopsy by Echeverria, sclerosis and amyloid degeneration of the antero-lateral columns, sclerosis of the anterior nerve roots, and brown pigment in the nerve cells, were found. In a case of von Recklinghausen's, tubercular deposit was discovered in the substance of the cord. H. Roger and Duchenne, Jr., reported two cases in which autopsy revealed atrophy of the anterior and antero-lateral columns, diminution in size of the nerve fibres, increase of the interstitial connective tissue, and the presence of numerous amyloid bodies. In an *unrecognized* case of infantile paralysis, Mr. J. Lockhart Clarke, of London, described an atrophy, a degeneration of the cells of the anterior horns.

To Prévost (a pupil of Charcot's), therefore, is due the credit of first recognizing this cell degeneration as the true lesion in this disease. In 1866 he published the report of the autopsy in which this lesion was discovered; since that date a number of cases have been reported by Charcot and his pupils, in which a similar cell degeneration has been found. Studied with the microscope, we find that it consists in an increase of the normal pigment of the nerve cells; the latter are observed to become densely packed with pigment granules; and finally, to wholly lose their cell character. In their place a simple granular mass, which gradually undergoes a marked diminution in size, is seen. In a certain number of cases no granular change is discoverable, the cells seemingly being subjected to a simple wasting process.

Other morbid changes indicating a myelitis, have been described as among the lesions of infantile paralysis, *e.g.*, a sclerosis, an increase of the interfibrillar connective substance of the medullary columns, with a marked increase in the nuclei of that substance; the formation of cavities in the gray matter, apparently through a process of liquefaction; small clots in the same substance; corpora amylacea in both the gray * and white matter; and finally what can only be described as condensed patches of tissue in the former.

The question is yet disputed whether the granular cell degeneration is to be regarded as the primary lesion. The fact that in one case at least † the cell lesion has been observed without any concomitant myelitis, would seem to support Prof. Charcot's view that the cell degeneration is the primary and essential lesion. This is further strengthened by the recent revelations concerning the state of the nervous centres in progressive muscular atrophy; in that disease the cells of the anterior horns being found in part or wholly destroyed by a similar pigmentary degeneration, without surrounding myelitis.

Prof. Charcot was so kind as to give me some sections of the cord from the case, now classical, which he published under his own name and Joffroy's (his interne) in 1870. I will pass around microscopes, each armed with a low objective, one showing a transverse section of a normal spinal cord, the other the section from Charcot's case. Both sections are from the mid-dorsal region of the cord, a part in which the anterior horns are small and the cells few. The normal anterior horn exhibits six or eight large, well-defined multipolar ganglion cells, stained red by carmine, their nuclei standing out more deeply tinged than the body of the cell. On the other hand, careful examination of the morbid section shows nothing but a somewhat condensed gray horn-tissue, without one distinct ganglion cell. The granulations which probably once existed in the place of some of the cells have been dissolved by the method (Clarke's) used in preparing the section. I would also call your attention to the fact, that the diseased anterior horn is shrunken and less club-shaped than the same part in the healthy section.

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* The theory now gaining ground in regard to the character of these bodies is, that they are due to an amyloid degeneration of the round cells of the neuroglia.

† Case by Charcot and Joffroy.

The DIFFERENTIAL DIAGNOSIS of this from other paralytic affections of infants I leave until our next meeting, when we shall review the whole group briefly.

The PROGNOSIS of infantile spinal paralysis is not good, for many reasons. The disease is a severe one, accompanied by a serious central lesion, and is possibly incurable in a certain proportion of cases in spite of every favoring circumstance. Then usually we are consulted weeks, months, and even years after the stage of atrophy and deformity has set in, and the cure is then more questionable in proportion to the period of time which has thus elapsed. Many of the deformities can be remedied, at any age, by proper orthopædic treatment; but that is not curing the disease. Until the recent (see *supra*) discovery of the reaction of atrophied muscles we had no guide in prognosis beyond time, and the appearance of the parts. Now we know that if any contraction can be obtained by means of the galvanic current (interrupted), there is some hope of restoring the muscles to activity. One authority (Dr. Hammond) * says: "If the muscles can be made to contract with either the induced or the primary currents, the cure is merely a matter of time and patience;" but I am afraid that this is rather a sanguine expectation. I should give a very guarded prognosis, under these circumstances, in all cases having lasted beyond a year.

I will be very brief about the treatment. The management of the first or febrile stage is a matter of uncertainty; few practitioners see the cases in this stage, and when they do, the diagnosis of simple fever, or of fever symptomatic of some teething or intestinal disorder, is usually made. Were I to meet with such a case, and have due reason, from occurrence of delirium, convulsions, and the presence of hyperæsthesia, to suspect impending spinal palsy, I should leech the child's spine, and apply counter-irritants to the extremities. Besides, treatment indicated by the state of the mouth or bowels should be carried out. The treatment of the second stage is likewise a matter about which no rules based on solid experience can be laid down. I should favor irritating the spine and the extremities, keeping the child's bowels free, and applying electricity, in either of the forms commonly employed, to the palsied muscles.

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* A Treatise on Diseases of the Nervous System. New York, 1871, p. 692.

The management of the third stage may be divided into (*a*) the treatment of the central lesion and of the atrophy; and (*b*) the curing of the deformities.

(*a*) We know no means which will with certainty remedy the central lesions which I have described as existing in this disease. Strychnia and nux vomica were prescribed by Heine and by other older authorities, but are now abandoned. The hypodermic injection of strychnia about the wasted parts has been recommended of late, and is worthy of a trial; from .001 to .004 may be injected with safety, according to the age of the patient. Of course everything which shall tend to improve the patient's general condition (hygiene, nutritious food, cod-liver oil, exercise) will favor the reconstruction of the atrophied nerve cells. The means of treating the palsy and atrophy consisted, until a few years ago, in the (nearly always vain) application of the faradic current, cold and hot douches, and the systematic friction of the atrophied muscles. These last are valuable, especially the alternate use of ice (or cold water) and hot water to produce hyperæmia. Beyond these in value comes the use of the galvanic current, which will in many cases produce good contractions in the wasted muscles. The number of cells to be used must be determined by trial, 10—20—30 elements of Stöhrer's battery may be required to obtain a reaction. The positive electrode should be placed upon the nerve-trunk supplying the atrophied group of muscles, and the negative sponge upon various muscles of the group; the current being meanwhile interrupted slowly by removing and replacing the negative electrode, or (to produce maximum irritation) reversed as well as interrupted by a mechanical contrivance on the battery or in the hand. I would suggest the use of nitrous oxide gas for the purpose of avoiding the intense pain produced by a large number of cells. Especially is anæsthesia useful in the first examination when you want to base a prognosis upon the result. An advantage offered by the use of this adjunct is, that the child being still, you are able to recognize a small muscular contraction which might be overlooked during the struggles of the suffering patient. How difficult it is to be sure that contractions occur under these circumstances, those of you who have attended in the electrical room of the college will remember. I am led to make a remark which I should have made when speaking of the prognosis, to the effect that a safe negative prognosis cannot in my opinion be based upon a first or second galvanic examination. If you can afford the time, and your patient the money, you should ask for a number of trials, at least six, before saying that the patient cannot be cured or improved. A great difficulty in the way of proper and successful galvanization consists in the very common stretched state of the atrophied muscles. For instance, in a palsied leg with healthy posterior tibial muscles, there exists a pes equinus, and in consequence the wasted anterior tibial muscles are stretched to a great degree. Now, gentlemen, I believe that this tense condition will prevent galvanic reaction for a long time, if not indefinitely; and the relief of the tension either by a mechanical contrivance (shoe), or better still by a tenotomy and a shoe, is followed by success in treatment. In illustration I would cite a private case of my own, a lady suffering from symptomatic muscular atrophy consequent upon cerebro-spinal meningitis, in whom the muscles of the legs were in much the same state as the muscles in cases of infantile spinal palsy; the posterior tibial muscles having in part recovered. The anterior tibial muscles were kept tense by a strong pes equino-varus, and for several months careful galvanization (even electro-puncture) produced no reaction. At last, as a *dernier ressort*, I asked Dr. H. B. Sands to cut the tendo Achillis in both legs, and to put the limbs in plaster of Paris. This was most dextrously done, and four days after the tenotomy distinct contractions appeared under galvanization in the atrophied muscles.

CASES OF INFANTILE SPINAL PARALYSIS.

NO	DATE.	AUTHOR.	AGE AT ONSET.	AGE AT AUTOPSY.	MODE OF ONSET.	PARTS PARALYZED.	LESIONS OF THE NERVOUS SYSTEM.	LESIONS OF MUSCLES.	BIBLIOGRAPHY.
1	1825	Hutin	7	49	Convulsions	Both legs	Atrophy of lower end of cord, and of its nerve-roots.	Not stated	Cited by Heine, Spinale Kinderlähmung, p. 151, 1860.
2	1842	Longet	..	8	Not stated	Right leg	Atrophy of roots of right sciatic nerve	Idem	Longet, Anat. et Phys. du Système Nerveux, I., p. 358. Paris, 1842.
3	1849	Fliess.	..	..	....	Upper extremities	Congestion of meninges over cervical enlargement of cord.	Idem	Cited by Laborde, Paralysie de l'enfance, p. 113. Paris, 1864.
4	1850	Rilliet	..	..	....	One arm	None. (Microscope not used.)	Idem	Gazette Méd. de Paris, 1851, p. 681.
5	1850	Idem	..	..	....	Both legs.	Idem. (Idem.)	Idem	Idem.
6	1855	Behrend	1	5	....	Right lower extremity	Chronic spinal meningitis.	Fibres small; not fatty.	Cited by Heine, op cit. p. 150.
7	1863	von Recklinghausen.	..	..	....	Both legs	Tubercles in cord	....	Deutsche Klinik, 31 Jan. 1863.
8	1862	Cornil	2	49	Unknown	Idem	Amyloid degeneration and atrophy of white columns of cord.	Fibres fatty	Gazette Méd. de Paris, 1864, p. 290.
9	1863	Bouvier	1	2	Idem	All limbs.	Sclerosis of antero-lateral columns of cord.	Fibres very small and granular; not fatty.	Cited by Duchenne fils in Archives Gén. 1864, II, p. 205 9.
10	1863	Henri Roger	..	..	Idem	Not stated	Idem	Not stated	Idem.
11	1864	Laborde.	8mo	2	Febrile	Both legs	Sclerosis of antero-lateral columns of cord; cells normal.	Fibres small; not degenerated.	Laborde, op. cit., p. 104.
12	1864	Laborde et Cornil	1	2	Idem	Idem	Cortical sclerosis of cord; sciatic neuritis; cells normal.	Fibres small and granular; not fatty	Idem, p. 109.
13	1865	Prevost	..	78	Unknown (infantile?).	Left lower extremity.	Atrophy of cells of left anterior horn of cord; atrophy of left antero-lateral column.	Fibres fatty	C.-R. Soc. de Biologie, 1865, p. 215.
14	1866	Echeverria	3	10	Febrile	Right limbs	Cells of anterior horns filled with granular pigment; diffused myelitis.	Fibres granular; not fatty.	Echeverria, On Reflex Paralysis, p. 29. New York, 1866.
15	1866	Idem	2	2½	Febrile diarrhœa	Both legs.	Nerve-cells granular; diffused myelitis; nerves shrunken.	Fibres fatty	Idem, p. 33.
16	1867	Wm. A. Hammond	..	..	Palsy of 4 yrs. standing.	Left lower extremity	Small clot in cord	Not stated	Journal of Psychological Med., 1867 p. 51. New York.
17	1868	Lockhart Clarke	1	32	After cow-pox inoculation.	Upper extremities	Atrophy of nerve-cells of anterior horns of cervical enlargement; central myelitis.	Idem	Med.-Chir. Trans., vol. LI., p. 249. London, 1868.
18	1870	Charcot et Joffroy	7	40	Sudden, non-febrile.	All limbs	Atrophy of cells of anterior horns; atrophy of antero-lateral columns; slight myelitis.	Fibres small, but not degenerated.	Archives de Phys., 1870, p. 135. Paris.
19	1870	Parrot et Joffroy	..	3	Unknown	Left lower extremity.	Atrophy of cells of left anterior horn; atrophy of left antero-lateral column, and nerve-roots.	Idem	Idem, p. 310.
20	1870	Vulpian	..	69	Idem	Right lower extremity	Atrophy of cells of right anterior horn; some sclerosis of white columns.	Fibres small; striated; finely granular (fatty?)	Idem, p. 316.
21	1871	Roger et Damaschino.	1½?	2	After dysentery.	Left upper extremity.	Atrophy of cells of l. ant. horn: sclerosis of antero-lateral columns; central myelitis and softening; l. ant. nerve-roots small.	Fibres small, with fatty granules and numerous sarcolemma nuclei.	Gazette Méd., 1871, p. 480.
22	1871	Idem	2	2½	After variola	Both legs.	Atrophy of nerve-cells of ant. horns; myelitis evidenced by sclerosis of ant.-lat. column and central spots of softening.	Fibres small; in part striated; with much granulo-fatty deposit.	Idem, p. 505.
23	1871	Idem	2	3	Febrile	Both legs and left side of back.	Atrophy of cells of ant. horns; p't'c's of softening in gray matter; sclerosis of ant.-lat. cols.	Idem	Idem, p. 541.
24	1873	Lancereaux et Pierret	2	18	....	Left arm	Atrophy of cells of l. ant. horn; atrophy and sclerosis of l. half of cord.	Not stated	Cited by Petitfils, Atrophie aiguë des cellules motrices, p. 33. Paris, 1873.
25		Roth	1	2	Febrile	Both legs	Atrophy of cells of ant. horns of lumbar enlarg., and cent. myelitis; atrophy of ant. nerve-roots.	Not stated	Virchow's Archiv. 1873; Bd. lviii., Heft, p. 263.

NOTE.—Dr. Allbutt's case (*Lancet*, 1870, II., p. 64), I reject because of evident traumatism.

NO.	DATE.	AUTHOR.	AGE AT ONSET.	MODE OF ONSET.	PARTS PARALYZED.	LESIONS OF THE NERVOUS SYSTEM.	LESIONS OF MUSCLES.	BIBLIOGRAPHY.
26	1873	Leyden, E..........	9 mos.	Not stated..	Left leg........	Scarcity and atrophy of the cells of the lanterior horn. Sclerosis in the antero-lateral columns.	Appearances of interstitial myositis.	Arch. für Psch. u. nerven krank., 1875, vi., p. 271.
27	1878	Taylor, F..........	15 mos.	Fever and pain (Blow)	Left leg........	Almost complete absence of cells in left anterior cornu. Those remaining are small, have few processes, and take staining fluids poorly. Also absence of axis cylinders in anterior cornu; gray matter of uniform, felt-like appearance; sclerotic changes in antero-lateral columns and marked atrophy of the anterior rootlets.	Not stated..........	Trans. Patholog. Soc., London, 1879, xxx., pages 197–215. Also Med. Times and Gazette, Feb. 15, 1879.
28	1878	Turner, F. C........	2 yrs.	Fall (?).....	Both legs, marked. Both arms, less marked.	Hemorrhage in the lumbar enlargement. Below the ant. cornua and outer part of base of post. cornua pale and translucent, and a confused appearance from leucocytal infiltration. Leucocytes massed together, especially in the perivascular sheaths of the smaller arterioles. Above the hemorrhage, scarcity of cells. In the antero-lateral columns young connective tissue cells in great abundance, and commencing sclerotic processes.	"	"
29	1878	Humphreys, Henry..	17 mos.	Enteritis....	Left leg........	Shrinking of left anterior horn, which contained few cells and axis cylinders.	"	"
30	1879	Bramwell, B........	15 mos.	Not stated..	Right lower extremity.	Destruction and disappearance of motor cells, which are replaced by fatty globules. Leucocytes in the anterior cornu, and throughout the gray matter. Atrophy of the anterior nerve rootlets and sclerosis of the anterior roots.	Of very pale tint.....	Edinburgh Med. Jr., 1880-1881, xxvi., p. 745.

Cases 26–30 are a few recent well-studied cases.—R. W. A.

A CASE OF TRAUMATIC BRACHIAL NEURALGIA TREATED BY EXCISION OF THE CORDS WHICH GO TO FORM THE BRACHIAL PLEXUS.[*]

WE offer the following case, believing it to be unique in its causation, and in the means used to relieve the terrible suffering caused by the nerve lesion.

History of the Case.—E. McA., an American, aged 18 years, was wounded in the following manner, at Worcester, Mass. On the 4th of July, 1871, he was aiding in firing a salute with a brass cannon. While he was ramming home the charge, standing on the right of the piece, his left hand by his side, and his right hand driving in the rammer, the piece was prematurely discharged. He was thrown a considerable distance (seven meters), and lost consciousness. In five minutes consciousness returned, and an examination showed no injury of any part excepting the right upper extremity, which exhibited a badly lacerated wound of the thumb and hand, a fracture of both bones of the forearm in the lower part of its middle third, and an extensive burn of the same part. Patient states in the most positive manner that his hand was absolutely without sensation at the time he regained consciousness, and remained "dead." Very shortly after the accident the metacarpal bone of the thumb was disarticulated, and as careful a dressing made of the fractured forearm as was allowed by the extensive burn. He was under the care of Dr. Albert Wood.

All apparently went on well until about three weeks after the accident, at which time pain showed itself in the range of distribution of the ulnar nerve of the injured side. In a few days this pain became constant, and assumed an extreme character, extending to the thenar eminence, and affecting the minimus and annulus fingers severely, the medius moderately; the pain was a cutting and tearing one. From that period neuralgia has been the predominant symptom, depriving the patient of rest, exhausting him physically, and quite breaking down his moral tone. Meanwhile, the wound resulting from the amputation of the thumb had partially healed, but no union had taken place between the fractured bones.

On the 11th of August the patient was brought to this city, and was attended by Dr. Salvatore Caro. Under this gentleman's care, narcotics, morphine, chloral, chloroform, were judiciously administered, but the controlling effect of these drugs was very fugitive ; the moment that the patient awoke from artificially produced sleep, the neuralgic pain in the hand and fingers reappeared with extreme violence, and caused the sufferer to groan and shriek. The wound caused by amputating the thumb rapidly healed, and the burn likewise cicatrized in greater part. From day to day the pain seemed, however, to grow worse, and the patient's strength and moral tone to fail. He became so irritable that the dressing of the wounds was a most difficult matter, inasmuch as he thought movement of the shoulder and arm increased his suffering.

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[*] By E. C. Seguin, M.D., in collaboration with Henry B. Sands, M.D. From the Archives of Scientific and Practical Medicine, Jan. 1873.

The operation in this case was planned by Dr. Seguin, tried on the cadaver by Prof. John G. Curtis, then demonstrator of anatomy, and performed with well-known skill by Prof. Sands. For a similar operation, see Drs. Maury and Duhring in the Am. Jr. Med. Sciences for July, 1874. [R. W. A.]

On the 14th of August, Dr. Caro called Dr. Sands in consultation, and the other author of this report was allowed by the courtesy of these gentlemen to take part in the examination. The following memorandum is a copy of notes taken shortly after the consultation by Dr. Seguin.

The patient is a well-built, muscular man, much emaciated. During the examinations he exhibits a truly extraordinarily nervous state, which his friends declare is quite unlike his usual manner. The face exhibits the traces of severe suffering and broken sleep. The right upper extremity is the seat of slight œdema. In the lower middle third of the forearm is a false joint, caused by the non-union of the fractured ulna and radius, the ends of which are considerably displaced. Nothing remains of the burn except a granulating surface, perhaps 3. cent. in diameter, on the inner surface of the lower third of the forearm. The amputation at the thumb-joint has left a small healthy oval ulcer. The entire upper extremity is motionless upon a pillow, the elbow and forearm being loosely supported by a simple felt gutter-splint. Patient fears that the examination will increase the intense shooting, burning and tearing pain which affects the fingers and the hand ; he dreads contact, active and passive motion. Consequently we are surprised to discover that (*a*) patient can make no voluntary motion of any part of the right upper extremity, except slightly raising the scapula, and that (*b*) sensibility is completely abolished as high as the upper part of the arm. The limit between absolute anæsthesia and sensibility is an irregular line which externally rises as high as the point of insertion of the deltoid muscle, and extends several inches lower upon the inner and posterior aspect of the arm. Above this irregular line of demarcation, about the scapular and clavicular regions, there exists great hyperalgesia, the patient complaining that the pain in the hand is excited by slight contact, and shouting and swearing from extreme agony when the scapula is handled. It is remarkable that bringing the scapula forward and holding it in this position gives the sufferer much relief. As regards the degree of anæsthesia existing below the above specified line—*i.e.*, in lower part of arm, entire forearm, and hand,—it may be stated that simple contact is not perceived ; that the fracture may be freely handled without causing pain ; that pushing pins deeply into the tissues is unnoticed; and that heated objects are not perceived.

From the patient's assertions about the effects of motion and contact upon the neuralgic pain, the belief had grown up that there might exist a relation between the symptoms of nerve injury and the fracture of the bones of the forearm; in other words, it was feared that the great nerves which pass among the muscles of the forearm to supply the hand were caught between the fractured bones, or were being compressed or irritated by fragments of bone. The question to be decided by the consultants, therefore, was the desirability of cutting down upon the fracture and determining whether any such pathological state as that above stated really existed. Of course, the discovery of the extensive paralysis and anæsthesia above referred to changed the aspect of the case materially. It was evident that we had to deal with an injury much higher up than the fracture, one producing a complete interruption of centripetal and centrifugal conduction in all the nerve-trunks which supply the upper limb. Of course, this being admitted as probable by all present, the conclusion was arrived at that no operation on the distal side of the injury could relieve the patient of his neuralgia; said neuralgia being a pain referred to the distribution of certain nerves, in accordance with the well-known physiological law of reference of sensations,—a pain whose cause was a nerve-lesion situated in the axillary space, if not higher.

Another consultation (Drs. Caro, Sands, Stephen Rogers, and E. C. Seguin) was held on August 15th, when the question of relieving the suffering of the patient was brought up. Dr. Rogers advised, with the view of interrupting the neuralgia, complete chloroform anæsthesia for a period of twelve hours. Dr. Seguin, considering the neuralgia as dependent upon the irritation of central ends of the injured (ruptured) nerves by newly formed (by repair) connective tissue, recommended counter-irritation to be applied near the supposed seat of nerve lesion, *i.e.*, above and below the clavicle.

On the 28th of August, Drs. F. H. Hamilton and Seguin were asked by Dr. Caro to see the patient. We find him in much the same state, suffering more, if possible; the pain being mainly of a burning character. The patient's excitement and irritability are such that details upon the state of his sensations are very difficult to obtain. The wound in the thumb has completely healed; but the fracture exhibits no signs of union. A careful examination of the state of sensibility shows that anæsthesia is complete in hand, forearm, and lower arm as high as limit indicated above. A new test is employed, viz., wire points connected with the maximum secondary current of a strong induction apparatus. Patient's general condition has somewhat improved. The existence of a severe nerve-lesion high in the axillary region (a rupture probably of all the nerves constituting the brachial plexus) being unanimously regarded as certain, and the chances of reunion of the torn nerve fibres and the regeneration of the peripheral parts of the nerves being looked upon as *nil*, it was proposed by Dr. Hamilton that the arm should be amputated near the limit of anæsthesia. It was thought (1st), that through the operation, some temporary alleviation of the neuralgia might be obtained; and (2d), that the patient would be rid of a member that would ever remain palsied and useless, and the care of which would interfere with the taking of exercise and with other means of regaining tone and strength. The proposal was concurred in; and, on the 29th, Dr. Hamilton dexterously removed the arm at about its middle, by the circular method. Very little blood was lost, and the operation was well borne. An experiment was made by Dr. Seguin upon the amputated arm immediately after its separation. A double-cell faradic battery was in readiness. The three great nerves, median, ulnar, and musculo-spinal, were rapidly laid bare at the upper part of the separated arm. They had lost their normal glistening, opaque appearance, and looked dirty and translucent. To these nerves, properly isolated upon glass, both the weakest and the strongest possible currents were applied without producing the slightest muscular contraction in the arm. The median and ulnar nerves were laid bare in the lower third of the forearm, examined in the same manner, and with the same negative result. Neurility, therefore, was abolished in these degenerated nerves, and a positive proof was obtained by this experiment of the correctness of the diagnosis of nerve-rupture. Contrarily, the muscles in every part of the extremity were found highly excitable, even feeble currents producing contractions. The interossei muscles, which respond least well, are infiltrated with serum, and are flabby and pale. A hasty examination of the arm showed the tendon of extensor carpi radialis torn across at its upper part ; no other muscles are injured. Muscles and tendons on ulnar side, opposite fracture, are covered with plastic exudations. The broken ends of the ulna and radius are not much displaced, but exhibit no trace of an attempt at repair. The nerves are in nowise involved in the fracture. The articular surface of the elbow-joint has lost some of its polish, and appears red.

One of us again saw the patient at the end of September. "I* learned that a degree of temporary relief had followed the removal of the limb. During the rest of the day of the operation only slight pain was complained of, but on the succeeding days it returned with increasing severity, until a fortnight later it was as great as ever, perhaps even worse. Patient has now completely lost self-control ; he swears frightfully, throws articles of furniture about, races up and down stairs in a five-story house, because of the intense burning, tearing and shooting pains which are referred to the hand and fingers. The worst times are in the afternoon and evening. Patient is then in a terrible state of nervous excitement ; he twists and squirms in his bed or chair, chews violently upon a handkerchief, and the perspiration pours from him. His language is interrupted by groans, oaths, and gnashing of teeth. Hypodermic injections of morphia—twenty and forty minims—with chloral, temporarily control pain. The appetite and nutrition have remained fair. Another consultation is proposed to be held between Drs. Caro, Hamilton, and myself. I am prepared to advise the section or resection of the nerves which go to form the right brachial plexus, at a point nearest the intervertebral foramina. The necessity for the performance of such an operation I base upon the diagnosis of injury (rupture) of the brachial plexus in the region where it is bound down to the vessels. I intend to cut the affected nerves above the seat of injury, and thus cause cessation of neuralgia."

The proposed consultation was never held. It was decided to try the controlling influence of a disciplined household upon his mental condition ; and he was accordingly sent the next day to the private institution for the insane under the charge of Dr. Barstow. The patient, let it be remembered, was absolutely sane; but it was thought that many of the new surroundings into which he would be thrown might strengthen his self-control and will to modify his expressions of agony.

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* Transcript from Dr. Seguin's memoranda.

On the 30th of October a brother of the patient called at my office, and stated that the family desired to place the patient wholly in my care; and he inquired what means, if any, remained, which would give a chance of relief from his great suffering. The operation above referred to was explained to him, and it was agreed that the trial should be made.

On the 2d of November the patient returned to town, and I visited him the same evening. He has changed very much for the better, his color having improved and his weight increased. He no longer cries out or swears because of the pain, but sits in a chair or lies in bed writhing, sweating, and chewing a handkerchief. The stump is of very good shape, and very nearly well. The neuralgia is still terrible, consisting mainly of shooting, tearing pains, together with some burning, and a sense of cramp in hand, all pain being referred to the extremity of missing member. Patient has been most judiciously treated by Dr. Barstow. He has had no morphia or chloroform for a month. He has eaten heartily, and has walked about a good deal.

An examination shows that the stump is sensitive, perhaps more so than is normal; the shoulder is much atrophied, and droops; the scapula is rotated by the action of the serratus magnus muscle. There exists some tenderness over nerves above the clavicle. The pain is continuous, with exacerbations in the afternoon, and during bad weather. Besides, he complains of his "hand feeling drawn up," and of "sinews working in the arm." With exception of constipation, no disturbance of any function is present.

November 5th.—The proposed operation is done by Dr. Sands. Present, Drs. Sands, Caro, Geo. A. Peters, Wm. H. Draper, F. N. Otis, T. T. Sabine, John G. Curtis, McCreery, and E. C. Seguin. Drs. Hamilton and Barstow had been invited to attend, but were unable to come. At 10.40 A.M. chloroform was administered, and anæsthesia continued by means of sulphuric ether. An ┘ shaped incision was made, its long arm extending parallel with the outer border of the right sterno-mastoid muscle, and its shorter arm following the clavicle. The flap was then raised and the connective tissue, with fibres of the platysma myoides and clavicular portion of sterno-mastoid muscle, divided and turned up. The external jugular vein was turned outward uninjured. Across the exposed triangle a vein larger than the external jugular was met with, apparently in very direct connection with the heart (showing systolic impulse), and, after being tied with two ligatures, was cut across. A little deeper the nerves were exposed without difficulty. It should be added that the latter steps of this dissection were done without cutting instruments. The connective tissue around the nerves did not separate with normal facility; the nerves constituting the brachial plexus were much matted together, and their dissection was by no means easy; still the first rib was plainly felt at the bottom of the wound, the scaleni were visible, and so was the anterior border of the right trapezius. The fifth, sixth, and seventh cervical nerves were cut in a lump, a piece fully a quarter-inch in length being excised; the same being done for a thick double cord, which seemed to represent the eighth cervical and first dorsal nerves. The pieces removed looked badly, and the nerves felt more like tendinous cords than like nerves. The surface of section appeared yellowish, showed hardly any trace of secondary fasciculi; and the neurilemma was unmistakably thickened and injected. More of the nerves (proximal ends) were taken away, Dr. Sands carrying his knife as near the scaleni as was practicable; but even there the sections exhibited the appearances of neuritis. During the operation no hemorrhage, worth naming, occurred. The carotid and subclavian arteries were both felt, but the phrenic nerve was not seen. Two or three very small arteries and the above-mentioned vein required ligature. The wound was closed by means of stitches placed .5 centimeter apart, drainage being allowed at the angle of wound.

This neuritis was not altogether unlooked-for by us. It may prove to be an inflammation which has ascended from the injured point, and which may be successfully treated afterward. Another possibility is, that the nerves have been cut below the seat of injury, in which case the neuralgia will return and persist.

Patient recovered from anæsthesia with much excitement and delirium; an hypodermic injection of sulphate of morphia .04 and .001 of atropia being administered before the ether effects had fairly passed off. After 1 o'clock P.M. he slept three hours. At 6 P.M. he is found rational, and moderately exhausted; pulse 120+, skin moist; has some headache; complains of soreness about shoulder, and of severe numbness in absent right hand, "just as when one's foot is asleep." Is chewing a handkerchief as before operation, though this is perhaps from habit. Ordered broths and a draught composed of 2. bromide of potassium and 2.6 hydrate of chloral, at 11 P. M.

9th.—A certain degree of pain returned after operation. Is quieted by hypodermic injections of morphia, .036, and atropia, .001. Some surgical fever.

10th.—No change in symptoms; a curious sore has appeared on the left ear. It is a superficial dry eschar, about a quarter of an inch square, on outer border of helix, on a level with tragus. Is this a reflex nervous nutrition disorder? Perspires more on right side than left; right brow wet, left quite dry.

26th.—Marked improvement. Numbness, with much burning, still present. Has taken KI 2. *per diem.* Has 1. cc. Magendie's solution of morphia at midday, 1.20 late at night, under the skin. Wound nearly closed.

Dec. 10.—Gaining. KI discontinued. Has lately taken quinia sulph. .30 twice a day; to be continued. Some dozen small blisters have been applied to various parts of the stump and shoulder with benefit. Has had exacerbation in changeable, stormy weather. Cigars have seemed to increase effect of morphia injections.

Jan. 1, 1872.—Last week passed through an attack of pneumonia (left lower lobe); defervescence in less than 48 hours. Neuralgia still severe, but decreasing. More self-control. Continue morphia under skin (1.5 to 2.6, in two doses), quinia; and ordered cod-liver oil.

April 1st.—The issue was closed about the middle of March; since has had a succession of blisters applied over stump and chest. Has much improved. Now sleeps in daytime and at night; gives much less expression to pain, although, in bad weather or during a change in the weather, he writhes somewhat, and perspires. The pain is of same character as at time of last note;

has much burning; very rarely any tearing or lancinating pain. Hyperæsthesia of skin of stump and chest continues. Fingers are still distinctly felt, and are the seat of most pain; the median and index appear glued together. Has noticed a curious associated sensation; which is, that whenever he squeezes strongly with the left hand he feels as if the absent hand were doing the same thing. There is much atrophy of muscles about right shoulder. The right pupil is smaller than the left;* and he sweats much more on the right than on the left side. General health is excellent, weight being 74. kilos.—greater than ever before. Receives injections at office, 1.1 to 1.2 (according to weather) in the morning (10 A. M.), and .72 to 1. about 7 P. M.

May 25th.—Since last note has improved in respect to neuralgia. Owing to the fact that he has not taken cod oil for some time, his weight has decreased some twenty pounds. The pain is nearly always burning; very rarely is there any shooting pain. The absent fingers appear to be in the same position as that detailed above. During the past three weeks he has observed more or less burning pain in cicatrix above clavicle; this pain is becoming daily more noticeable. He has also suffered somewhat from end of the stump. Has regularly received hypodermic injections of morphia night and morning, 1.1 and 1. of Magendie's solution. An examination of the stump and shoulder is made to-day. These parts are very much atrophied, the acromion and coracoid processes being quite prominent. The scapula has rotated outward and upward in such a way that the acromion process is raised, the posterior border of the scapula drawn away from the spinous processes of the vertebræ, and the inferior angle made to approach the axilla. There is no tendency to the "wing" deformity; *i.e.*, the serratus magnus muscle is not paralyzed. This one and the muscles raising the scapula (trapezius and levator anguli scapulæ) are the only muscles of the region which have escaped atrophy. Forced chest-expansion is very good on both sides. There exists a lateral spinal curvature in the lower cervical region (convexity toward the injured side), and another in the opposite direction (compensatory or result of pneumonia ?) in the lower dorsal region. The end of the stump is very firm and sound; the cicatrix above the clavicle is also in good condition. State of sensibility.—The patient states that he has an extensive surface on the right side that is abnormally sensitive. Light contact and pinching are felt a little less distinctly on this zone than on the corresponding parts of the left side; the æsthesiometer test reveals no difference between the two sides. Cold is perceived a little more distinctly on the left side than on the right. While light contact and pinching are less acutely felt in *the part touched* on the right side, these same irritations (and any others) start the neuralgia with a severity proportioned to the acuteness of the impression. This falsely hyperæsthetic region has the following limits : The entire stump and shoulder; the scapular region, and a little of the back inside of and below the scapula; the axillary region, and the pectoral region as low as a point one inch below the nipple; the inner anterior limit is along the right outer edge of the sternum up to the supra-sternal notch, where the limit extends quite to the median line, thence taking an oblique course along the anterior edge of the sterno-mastoid muscle, then a little forward so as to include the angle of the jaw and a part of its ramus and body; from the lobule of the ear the line extends backward and downward to the posterior angle of the scapula. The teeth on the right side have been so sensitive that he has not brushed them for months; nor has been able to comb his whiskers on that same side. We repeat that this abnormal sensitiveness is not a true one, not in the parts touched or pinched, but that irritation of this zone excites the neuralgic pains, these being of the nature of associated sensations. The pupil on the side of the injury and operation is distinctly smaller than that on the sound side (left). The perspiration is more abundant, and appears more quickly upon the right side than upon the left. During the examination the left axilla was moist, but two or three large drops of sweat tickled down the side from the right.

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* This disparity in the state of the pupils was seen very shortly after the operation, but no note made of it.

During the last two months the neuralgia has been much less influenced by changes in the weather. It is decided to try applications of the actual cautery to the shoulder and chest. Choice is made of the platinum-tipped cautery applied at white heat, and in a superficial way (Brown-Séquard's method). Morphia to be continued.

June 22d.—The cautery was applied in all some five times without producing any noticeable relief. Pain is severe, but patient has some hours of sleep, and others of comparative ease, while taking only .80 Magendie's solution night and morning. Neuralgia presents same character, consisting mainly of burning, referred to fingers. These last seem to be in peculiar position above described. Patient is left for the summer under the supervision of Dr. A. Brayton Ball.

December 1st.—Since last note no marked change has occurred. Patient still suffers from much burning and from some lancinating. This is, as of old, referred to fingers and hand, being felt slightly and seldom in stump or supra-clavicular cicatrix. Hallucination regarding position of fingers continues the same. He thinks he has had more pain in last two months, but this is to be judged in connection with the fact that the morphia has not been increased; takes .80 Mag. night and morning. Pupils are still unequal. Right side (same parts) still exhibits false hyperalgesia, less marked. Has lately combed whiskers and cleaned teeth on that side. Still perspires more on right side. Right side of neck and other parts have been irritated. and no epileptiform symptoms produced. General health good.

There are a number of points in this history which, we believe, require more extended consideration.

1. The pathological anatomy of the nerves involved. At the time of the amputation, portions of the three great nerves of the arm, median, ulnar, and musculo-spiral, were removed within two hours after the separation of the limb, and immersed in a weak solution of chromic acid. Two or three weeks later, transverse sections were made of these nerves, and treated in a way to be subsequently described.

During the operation performed by Dr. Sands, on November 5th, pieces were removed from the cervical nerves and from the dorsal nerve which go to form the brachial plexus. These pieces, varying in size from .5 to .75 centimeter in length, were cut off as near the scaleni muscles as it was possible to carry the knife. As related in the history of the case, these fragments and the nerve-trunks from which they were taken looked wholly abnormal. The connective tissue surrounding them was hardened and thickened, the nervous cords no longer appeared pearly white or glistening, and the surfaces of section showed no trace of secondary fasciculi and no attempt at breaking up into bundles, as are seen when a normal nerve is cut across. These fragments were also immersed in dilute chromic acid, and when they were hardened transverse sections were cut from them. These sections, and those from the nerve of the arm, were stained by neutral carmine solution, the water removed from them by successive washings in alcohol and absolute alcohol. They were then made transparent by being floated upon oil of cloves, and finally mounted in Canada balsam dissolved in chloroform.

Before proceeding to the description of the alterations presented by these sections, it may be well to give a cursory account of the appearance of a normal nerve section prepared by the same (Clarke's) method. In section of a normal sciatic nerve* seen with a power of 65 diameters (see Pl. I.), Fig. 1 exhibits every nerve fibre as a little circle, within which is a hyaline mass, and in the midst of this mass a red dot placed a little to one side of the centre in most cases. These parts are the axis cylinder as the central dot, the white substance of Schwann or myeline as the hyaline mass, and the membrane of Schwann as the circle or rounded ovoid. As shown in the figure, these circles (varying a little in diameter) crowded together constitute the secondary nerve bundles or fasciculi, which are so large that most of them are clearly seen by the unaided eye. Between the nerve fibres is a uniting substance which appears faintly striated; and here and there are stronger bands of connective tissue (trabeculæ) which are united with the connective tissue around the fasciculus. This is shown (*b*, *b*′, Fig. 1) as a thick ring, apparently made up by the aggregation of nearly parallel fibrillæ. Around each secondary fasciculus of a spinal nerve there is such a sheath appearing as a ring in transverse sections; and these sheaths are united among themselves by more or less loose connective tissue (*b''*, Fig. 1). In this loose connective tissue run blood-vessels (*c*, Fig. 1), arteries, and veins of various calibre. There are small additional blood-vessels enclosed in the perifascicular sheath and in the delicate tissue which separates the nerve fibres.

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* All spinal nerves present essentially the same appearance.

a. The changes exhibited by the median, ulnar, and musculo-spiral nerves. To the naked eye sections of these nerves show traces of secondary fasciculi, although the picture is far inferior to that seen in the normal section. Under a power of 65 diameters the connective tissue around the nerve and that between the secondary fasciculi appears moderately increased in quantity and density. The perifascicular sheaths themselves have lost their definite outlines, and merge more into the connective tissue lying round about them. The sections of blood-vessels seem but little changed, and only a few granular (yellow) bodies are seen in the interfascicular tissue, mainly in the neighborhood of the vessels. The great alteration is in the nerve fibres. In the fasciculi very few distinct circles are to be seen, the mass constituting the fasciculi appearing as a confused design made up of fragments of circles heaped one upon the other. In none of the remaining circles can an axis cylinder be satisfactorily recognized. In such circles as exist, the hyaline substance within them (myeline) appears more refracting than is usual, and is often concentrically striated. We have here the lesions characteristic of the Wallerian degeneration, *i.e.*, disintegration of the nerve fibres, with proportionately little change in the framework of the nerves.

b. The sections from the nerves excised on November 5th. These present an altogether different appearance. To the unaided eye they appear like sections of some dense, indistinctly fibrillated tissue, tendon for example. Under a low magnifying power the general sheath of the nerve is seen very much hypertrophied. The secondary fasciculi vary immensely in size and appearance. A few are still rounded, encircled by a distinct sheath, and fairly filled with nerve fibres in better or worse condition. The majority, however, are broken up into innumerable smaller bundles, the separation being effected by the formation of distinct bands of fibrillated connective tissue in the place of the scanty network described as lying between the fibres in a normal section. Between many of these fragmented fasciculi are huge masses of waving, dense connective tissue, with abnormally large vessels, and with a great quantity of granular pigment deposit. This yellowish pigment lies principally immediately around the blood-vessels, or in the connective tissue near them.

As regards the nerves themselves, it may be stated in general terms that they are in a state of atrophy. In one fasciculus, for example, there are very few fibres which present the circular outline, hyaline mass, and central dot characteristic of the normal fibre seen in transverse section. The vast majority are much smaller than usual (appearing of about the same dimension with 300 diameters as normal fibres do with 65); they vary immensely in diameter, and many are represented only by parts of small circles. No masses of embryonic cells are seen in any part of the preparations. Fig. 3 is drawn from a preparation made from the eighth cervical and first dorsal nerves under a power of 300 diameters, and exhibits very fairly the condition existing in one of the best preserved parts of the section. One large fasciculus is quite entire, though the majority of fibres composing it are shrunken and empty. Above this fasciculus are very heavy masses of connective tissue, which is closely connected with the perifascicular sheaths. To the left of this fasciculus is seen a blood-vessel having round about it much yellow granular pigment. The fasciculus represented in the left upper part of the sketch is broken up by increase of the connective tissue into tertiary fasciculi, and many smaller aggregations of atrophied nerve fibres; in some places single fibres are seen surrounded by dense connective tissue. In other parts of the preparation more extreme changes are to be seen, in some fasciculi nearly every fibre being separated from its neighbors by newly formed fibrillar substance.

To resume: The nerves in the upper cervical region present the lesions characteristic of chronic neuritis, viz., much increase and condensation of the framework, with comparatively minor change in the nerve fibres. In other words, the pathological process in these nerves has been primarily hyperplastic, and the neural atrophy secondary and incomplete; whereas, in the nerves removed from below the axillary space, the neural atrophy was complete and primary, the changes in the framework very slight.

In the one case we have the lesions of chronic hyperplastic neuritis; in the other, those of the Wallerian degeneration.

2. Nature and seat of the injury to the nerves.

The absolute anæsthesia of nearly the whole arm exhibited by the patient previous to its removal, and which probably existed immediately after the infliction of the injury, points to a complete solution of continuity in all the nerves which supply the lower arm, forearm, and hand with sensory filaments.[*] Further, the patient had complete paralysis of muscles situated far above the limit of anæsthesia, those which act upon the upper part of the humerus and some of those moving the scapula. The distribution of motor palsy and of anæsthesia in this case fully illustrated van der Kolk's law of distribution of sensory and motor filaments of one nerve trunk, viz., that the former are sent to parts which are moved by muscles innervated by the latter.[†] We therefore had ample clinical reasons for localizing the injury at least as high as those parts of the brachial plexus which lie behind and just above the clavicle, and also for considering that the injury consisted in a complete disruption of the nerve trunks. Another possibility presented itself to our minds, viz., the tearing out of the roots of the nerves which constitute the brachial plexus from their attachment to the anterior and posterior aspects of the spinal cord. Such an accident has been placed on record by Flaubert,[‡] occurring as a consequence of forced extension made to reduce an old dislocation; but in this case the patient died in a few days with symptoms of inflammation of the spinal cord, corroborated by the autopsy. Guided by the result of this case, and by the fact that our patient had at no time presented any symptom of spinal meningitis or myelitis, we felt reasonably certain that the nerve roots in this case had not been torn out. Having thus excluded intra-spinal rupture and determined with certainty the lowest possible limit of the injury, the question arose, whether we could arrive at a still more exact knowledge of the seat of nerve rupture; where in this tract between the intervertebral foramina giving issue to the fifth, sixth, seventh, eighth cervical, and to the first dorsal nerves, and the upper limit of the axillary space, was the laceration most likely to have taken place? It appeared to us impossible to make a satisfactory answer to this question. The microscopical examination of the nerves corroborated the diagnosis reached upon clinical grounds, since the sections taken from the upper part of the cervical nerves showed neuritis, while those cut from nerves below the axilla exhibited the changes of descending or Wallerian degeneration. It is therefore right to conclude that the excision has been made, as intended, above the seat of laceration.

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[*] Compare Mitchell, Injuries of Nerves. Philadelphia, 1872; p. 227.

[†] Schroeder van der Kolk. On the Minute Structure and Functions of the Spinal Cord and Medulla Oblongata. Translated by the New Sydenham Society. Vol. iv., 1858; pp. 8, 9.

[‡] Répertoire Général d'Anatomie et de Physiologie Pathologique. Vol. iii., p. 55. Cited by Le Bret, Mém. de la Soc. de Biologie, 1853, p. 121.

In this connection it may not be amiss to recall the exact mechanism of the accident. The patient's right hand was firmly clasping the rammer, and all the muscles of the arm were in activity during the effort of ramming home the charge. The explosion naturally drove the hand forward and outward with incredible violence, the arm following the same direction, and being for the moment in a state of extremely violent extension. So enormous was the strain upon this limb that the patient was thrown bodily quite a distance. We see no reason for not admitting that the fracture of the bones of the forearm occurred at the beginning of this movement of extension. This being granted, it follows that a great strain was put upon the soft parts which still connected the lower part of the forearm with the upper, and that the blood-vessels and nerves were greatly elongated. During this elongation the nerves gave way at their weakest point, *i.e.*, where they are most firmly bound down, and where they interlace and anastomose—behind the clavicle.

Besides Flaubert's case above referred to, we have met with quite a number of instances of obscure nerve injury caused by the reduction of old shoulder dislocations, but the details given are so meagre as to make the cases quite useless. An exception to this statement is the case recorded by Le Bret.* A young soldier, who had dislocated his right shoulder, underwent the operation of reduction on the same day. The traction was done by men pulling upon a sheet firmly tied around the arm just above the elbow. Immediately after the reduction, without any special pain having been felt, the patient noticed that his arm and forearm were paralyzed. When seen by Le Bret, five months later, there existed complete anæsthesia below the bend of the elbow, besides palsy of the arm. The corresponding side of the neck had lost motion, and was anæsthetic; the right upper eyelid covered the globe, and vision was impaired; the right iris was slightly contracted. There were some lancinating pains in fingers and arm. The nerves (inner aspect of arm, and above clavicle) were tender to pressure. Some improvement took place in motion of arm and neck, and the ptosis was cured. The author believes that the nerves were torn across in the region of the brachial plexus.

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* Mémoires de la Soc. de Biologie de Paris, 1853, p. 119.

3. The demonstration of persistent muscular irritability at a considerable period after the muscles had ceased receiving nervous influence.

The arm was removed eight weeks after the reception of the injury, and, as related above, while no muscular contractions could be obtained by faradizing the nervous trunks at various points, almost normal movements were produced by the direct application of the current to the muscles themselves, even those (interossei) which had apparently suffered much in their nutrition. The bearing of this experiment upon the question of the independence of muscular irritability might detain us awhile, were it not that this paper has already reached a considerable length. Let it suffice to state that this result agrees with that obtained in the inferior animals. The fact that functional capacity survives in muscles for a period six or twelve times longer [*] than in nerves, in cases where those organs have been cut off from communication with the spinal cord, has been demonstrated by a great number of physiologists. Among the earlier of these we may name Marshall Hall, J. Müller, Günther and Schön; the latter fixing the date of loss of excitability in nerve trunks at eight days after section. Later experiments by Longet, Schiff, Landry, Vulpian, and many others have resulted in positively limiting the time at four days. On the other hand, these observers agree in stating that muscles retain for a much longer period the power of reacting under immediate stimuli. Some of Longet's [†] conclusions on this point are worth reproducing:

"1. In mammals, a motor nerve, when separated from the cerebro-spinal axis, loses all excitability after the fourth day. At that time the application of mechanical, chemical, and electrical irritants to any part of the distal end of the nerves is followed by no muscular contraction.

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[*] Dr. Brown-Séquard asserts that there is sometimes no diminution of muscular irritability: he has found it as great as in the normal state nineteen months after the whole central end of the facial nerve has been drawn out from its exit at the stylo-mastoid foramen. Bulletin de la Société Philomathique, 1847, p. 83.

[†] Traité de Physiologie, t. ii. p. 619. Paris, 1869.

"2. Contrarily, a muscle whose motor nerve is no longer excitable, will, even after the lapse of twelve or more weeks, respond perfectly to any direct stimulus."

Landry,* however, states that in the human species, muscular irritability under these circumstances is abolished in the seventh week. The almost perfect response of the muscles to stimuli in our case, and their apparently normal structure at the end of eight weeks, completely overthrows Landry's conclusion. The causes of error in the author's observations lay, 1st, in the fact that he was unable to apply the electric current directly to the muscles, although he made use of electro-puncture; and that, 2d, in all likelihood there existed in his cases more or less active impairment of nutrition in the paralyzed muscles, owing to irritation of the nerves at their origin.

Vulpian† rightly insists upon the value of the fact observed by him in animals, that muscles deprived of innervation which do not contract when the electric (faradic?) current is made to pass through the moistened skin, do so fairly when the electrodes are placed immediately upon the muscular substance; and he goes on to throw doubt upon the observations made by clinicians in regard to the early (fourth—eighth day's) loss of electro-muscular contractility in certain palsies—the "rheumatic" paralysis of the face, for example. It is to be regretted that in our case the patient's great suffering deterred us from faradizing the muscles of the arm before its amputation.‡ As it stands, our observation is in favor of a prolongation of muscular irritability in man after nerve section for a period quite as long as that determined in the lower animals.

4. Some of the symptoms appear to us especially interesting.

(*a*) In the first place, there are signs pointing to a paresis of the vaso-motor nerves on one side of the face, neck, and chest. The right pupil was noted as smaller than the left immediately after the operation, and from an early period the patient perspired much more upon the right side than the left. Besides, there was a peculiar condition of sensibility on a large extent of the right side of the body. At one period this is spoken of as hyperalgesia; but a later examination showed that there was no abnormal tenderness in the part touched, and that the pain produced by contact was felt in the absent arm and hand. Still, it should be borne in mind that the patient's self-control and estimation of the nature of his sensations were not always normal, so that it cannot be asserted that there did not exist, at an early period, true hyperalgesia. The æsthesiometer certainly taught us nothing. The extensive surface, falsely sensitive, bore the same relation to the brachialgia that many "tender points" do to ordinary neuralgia. An impression transmitted to the spinal cord, at a point near the portion which gives origin to the nerves supplying the region affected with neuralgia, causes action of the sensory tract connected with these nerves, and consequently produces a *referred* sensation of pain. One of us * had occasion to observe a curious example of this *associated painful sensation* in his own person, last summer. A lower incisor tooth had become the seat of tartar deposit, and the gum below was shrunken, red, and tender to the brush. There had never been toothache. One day a small pimple appeared on a level with the upper margin of the thyroid cartilage on the same (right) side as the unhealthy gum; and during the entire period of growth and maturity of the pimple, pressure (even light) upon it produced an acute pain in the gum around the above-mentioned tooth. The experiment was repeated scores of times; and it was further observed that touching the gum did not produce pain in the pimple. Here an impression made upon a branch of the superficial cervical plexus, transmitted to the sensory tract of the upper cervical cord and medulla oblongata, excited in the latter action of the cells connected with the third branch of the trigeminus.

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* Traité complet des Paralysies, t. i. pp. 40–41. Paris, 1859.

† Leçons sur la Physiologie du Système Nerveux, p. 245. Paris, 1866.

‡ Through causes beyond our control, the galvanic current could not be applied to the nerves and muscles in the above-detailed experiment.

(*b*) The disturbance of nutrition, which produced a slough upon the left helix, is difficult of explanation. It is well to remember in this connection that Brown-Séquard produces gangrene of the edges of the external ear, at will, in guinea-pigs, by injuring the medulla oblongata.

(c) The burning pain (causalgia of Mitchell) did not appear immediately after the injury; this being in accordance with the rule laid down † by the distinguished author just named. As regards the date of the appearance of this peculiar pain, we can obtain no definite information.

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* Dr. Seguin.

† Mitchell. Injuries of Nerves, and their Consequences, p. 197. Philadelphia, 1872.

The expressions of agony, in action and words, employed by our patient corresponded singularly with those recorded by Dr. Mitchell in his works upon nerve injury.*

With reference to the extraordinary severity and persistence of burning pain in cases of injury to nerves, we would recall the fact, first distinctly stated by Cruveilhier,† that loss of the power of perceiving thermal impressions occurs later than the loss of various other varieties of sensibility, and indicates absolute anæsthesia; and we suggest that inasmuch as the thermal sense is the last to disappear in gradual diminution of sensibility, so in a neuralgia caused by irritation of nerve trunks, this most deeply rooted, or most fundamental mode of sensation is most affected, and burning is felt acutely when common pain and formication have almost or quite ceased. It is well known that extreme irritation of the skin, after producing ordinary pain, causes intense burning; an event frequently met with in surgical practice; and, moreover, the contact of extremely cold bodies with the skin sets up a painful sense of heat.

5. The operation above described is believed to be the only one of its kind ever attempted. Excision or division of the spinal nerves has generally been performed on the smaller branches; and, excepting the case herewith related, has never involved the primary trunks near their points of exit from the spinal canal. Neurotomy, when undertaken for neuralgia of traumatic origin, has, in a great many instances, effected a permanent cure, and in these cases is far more likely to prove successful than when it is performed for the idiopathic forms of the disease. If the nerve tissue is healthy at the point of section, the operation can hardly fail; yet success has followed the operation in not a few cases where the divided nerve was thickened and inflamed. In the lower extremity, excision of the smaller nerves has repeatedly been performed, and in several instances, the great sciatic has been either excised or divided. Dr. Mitchell ‡ reports a case in which Dr. Nott excised 3. centimeters of the great sciatic nerve, close to its point of exit

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* Compare, also, Mitchell, Morehouse, and Keen. Gun-Shot Wounds and other Injuries of Nerves. Philadelphia, 1864.

† Anatomie Pathologique, liv. xxxviii. p. 9.

‡ S. Weir Mitchell, op. cit. pp. 285, 286.

from the pelvis, for traumatic neuralgia caused by a gunshot wound of the leg. Amputation of the leg, reamputation of the stump, excision of the sciatic nerve in the popliteal space, and amputation of the thigh, had already been performed in succession without avail. Partial relief is stated to have followed the final operation performed by Dr. Nott.

Other cases of division of the great sciatic nerve are recorded by Malagodi, Mayor, Nélaton, and Jobert de Lamballe. In Jobert's case the operation was performed for sciatica. Pain ceased at once; but death occurred six months subsequently from paralysis and bed sore.

In the upper extremity, excision of the median and several other branches of the brachial plexus has often been practiced, and with various results.[*] In some cases the operation has effected a complete and permanent cure; while in others it has afforded no benefit. Several years ago one of the authors[†] treated a patient in Bellevue Hospital who suffered from violent neuralgia and chorea, caused apparently by a neuroma which had formed upon the face of a stump after amputation of the arm near a shoulder joint. The neuroma was laid bare by dissection and was found to be connected with all the descending cords of the brachial, excepting the circumflex. These were pulled downward, and, together with the auxiliary vessels, divided at about an inch above their seat of attachment to the neuromatous swelling. The neuralgia was relieved by the operation while the patient remained under observation, but the choreic symptoms persisted. He left the hospital about two months after the operation.

In the case which forms the subject of this article, the operation of excision of the spinal nerves was undertaken partly as a last resort, and partly because it was thought that the danger of performing it would be considerably reduced, in consequence of the previous removal of the arm by amputation. It is interesting to observe, however, that no serious nutritional changes, except those affecting the muscles, took place in the parts supplied by the divided nerve trunks.

Another point of interest is the practicability of the operation when considered merely with reference to the difficulty and danger attending its execution. Under ordinary circumstances, supposing the nerves to be healthy near the points of section, the operation would cause no embarrassment to a skilful surgeon, and all the cords of the plexus might be exposed and divided without dangerous interference with the neighboring blood-vessels. But, even in the present case, where the nerve trunks were pretty firmly adherent to the surrounding tissues, their isolation was satisfactorily accomplished by careful dissection; and the wound made by the operation healed readily, without profuse suppuration.

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[*] Schmidt's Jahrbücher, cxxii. p. 218; Bd. cxiii. p. 298 *et seq.*

[†] Dr. Sands.

Examination of the nerves excised led to the unsatisfactory conclusion that they were diseased above the line of section; and it is not easy to understand, on anatomical grounds, why any benefit should have followed the operation. The nerve trunks, however, were divided pretty close to the intervertebral foramina; and, if it be assumed that the cause of pain resided in their proximal ends, it is not improbable that the tension of the latter may have been diminished, and their relations otherwise favorably altered as a consequence of the handling to which they were subjected previously to their division. Such an explanation seems plausible from the results that attended an operation recently performed by Professor von Nussbaum, an abstract of whose paper appears in the present number of this journal. It may also be supposed that the cutting off of a considerable portion of irritated nerve trunk from communication with the spinal cord diminished the neuralgia by reducing the total amount of irritation transmitted to the nervous centre.

We may sum up the case by stating that a neuralgia of a class known to resist all ordinary treatment was much relieved by an operation not dangerous in itself. We did not obtain radical success, because we failed to find healthy nerve trunks at the place of section. The diagnosis of the seat of injury was correct enough, but the ascending neuritis baffled us.

We are indebted to Dr. Caro for a statement of the case as it appeared to him, but as his letter contains nothing that is not recorded in the above history, we take the liberty of omitting it.

EXPLANATION OF PLATE I.

FIG. 1. Transverse section of fasciculus of normal spinal nerve.

a, Nerve fibres seen in section, exhibiting circle (membrane of Schwann), hyaline contents (myeline), central dot (axis cylinder).

b, Sheaths of secondary fasciculi.

b', Trabeculæ of connective tissue which subdivide fasciculi.

b'', Interfascicular connective tissue.

c, Sections of blood-vessels.

FIG. 2. Fasciculus from ulnar nerve, in middle of arm, showing the lesions of Wallerian degeneration.

a, Confused outlines of nerve fibres, caused by loss of myeline and axis cylinder, and collapse of membrane of Schwann. Not one axis cylinder is to be seen.

b and *b'*, Sheath of fasciculus, and interfascicular areolar tissue, a little thickened.

c, Unaltered blood-vessel.

FIG. 3. Section from lower cord of brachial plexus near intervertebral foramina, showing the lesions of chronic neuritis.

a, Secondary fasciculi, showing atrophied fibres (circles not much larger, under three hundred diameters, than those of normal nerve under sixty-five diameters); very few axis cylinders present. Tissue between fibres increased.

a', Small aggregations of fibres, separated from others by dense inter-fibrillar connective tissue.

b, Immensely hypertrophied interfascicular areolar tissue. Sheaths of fasciculi no longer distinct.

c, Dilated blood-vessels surrounded by altered connective tissue.

d, Yellow granular pigment lying in areolar tissue, mostly in neighborhood of vessels.

The various specimens were prepared according to Clarke's method.

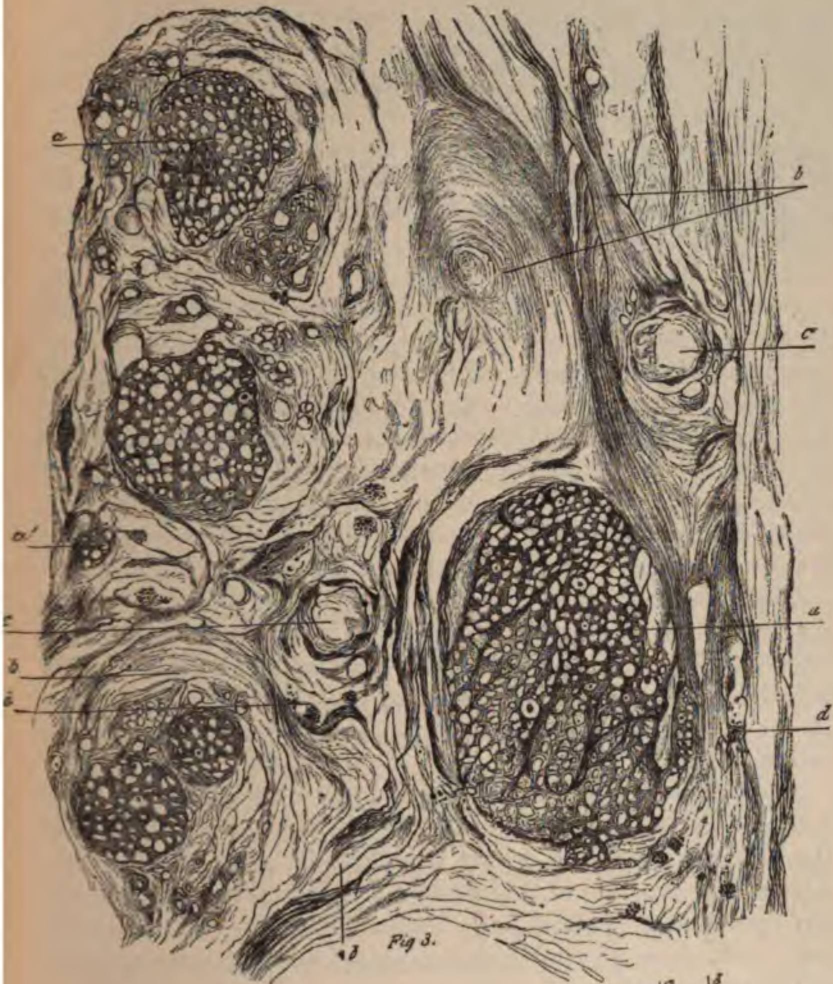

Fig 3.

.1mm × 300.

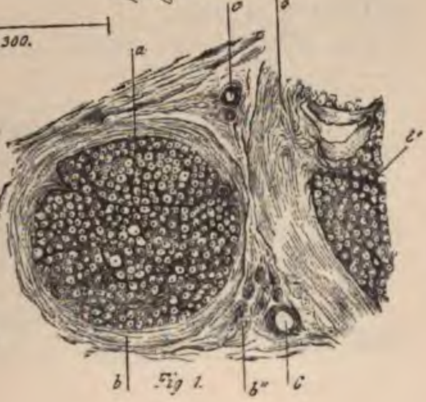

Fig 1.

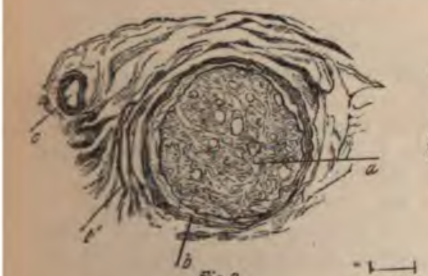

Fig 2.

.1mm × 65.

ON THE INHIBITORY ARREST OF THE ACT OF SNEEZING, AND ITS THERAPEUTICAL APPLICATIONS.[*]

In the early summer of 1869 I discovered that sneezing could be arrested or prevented by forcibly rubbing the skin below and to either side of the nose. I at once communicated this fact to the editor of this Journal, and in my letter pointed out the inhibitory nature of the phenomenon; classing it with the arrest of the heart's action in Weber's experiment,[†] and with the arrest of spinal epilepsy by irritation of the great toe.[‡] I was soon made aware that this simple means of stopping sternutation was known to many outside the profession, and that it had been formally recommended by Diday[§] in 1843. More recent researches have shown me that Haller[‖] (perhaps Bartholinus before him[¶]), and Marshall Hall[**] had called attention to the possibility of arresting sneezing by irritating the skin between the nose and the angle of the eye (Haller), or by rubbing the end of the nose (M. Hall).

I venture to treat formally of this small matter, because I was perhaps the first to call attention to the correct explanation of the arrest, and because I have long had in mind certain therapeutical applications of this method. Before proceeding to state the circumstances which may indicate the arrest of sneezing, a few words about physiological and pathological sternutation may not be out of place.

1st. What is the physiology of sneezing? A most distinguished physiologist* has said that "sneezing is a sudden and violent contraction of the expiratory muscles consequent upon the closure of the upper part of the air passages. * * * The diaphragm, contrarily to what is taught by many ancient and modern writers, takes no part in the act of sneezing; it is not an expiratory muscle, and acts only in the deep inspiration which precedes sneezing." Austin Flint, jr.† defines sneezing as "a convulsive action of the expiratory muscles succeeding a deep inspiration; the air being violently expelled, with a characteristic sound, through the nares." Carpenter ‡ states that the act of sneezing is accomplished by a violent expiratory effort. Haller § speaks of sternutation as consisting of a spasm of the diaphragm in the first place, causing a deep inspiration, this being followed by spasmodic action of the muscles of expiration. Morgagni ‖ also considers the act of sneezing as caused principally, if not wholly, by spasmodic action of the diaphragm. Many writers mention the associated movements of the face, head, neck and limbs, which every one must have observed in his own person while sneezing. After the deep inspiration, the air is held in the chest by closure of the glottis, then escapes principally through the nose, the isthmus of the fauces being closed. This is the typical sneeze; but custom has brought about the discharge of a part of the air through the mouth, in order to prevent the very result for which the act of sneezing seems fitted, viz., the expulsion of mucus from the nostrils. I desire particularly to call attention to the Hallerian view of the physiology of the act. It appears to me that the deep inspiration which we take previous to sneezing is really spasmodic, and due to a morbid (unusual) irritation of the centre of origin of the phrenic nerves. In support of this ancient view we have the fact that in cases of excessively prolonged sneezing (Brown-Séquard's and Mosler's cases) the pain complained of is located in the neighborhood of the diaphragmatic insertion. Could we prevent this first or preliminary act, we should always be able, it strikes me, to avoid sneezing.

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[*] From the Archives of Scientific and Practical Medicine, March, 1873.

[†] See Brown-Séquard, Archives of Scientific and Practical Medicine, No. 1, Jan., 1873, p. 89.

[‡] Brown-Séquard, Archives de phys. normale et pathologique, i. p. 157, 1868.

[§] Diday, Note sur un moyen simple de prévenir ou d'arrêter la toux dans certaines maladies. Gazette Méd. de Paris, 1843, pp. 103–106.

[‖] Haller, Elementa physiologiæ, t. iii. p. 304. Lausanæ, 1766.

[¶] Th. Bartholinus, cited by Haller *in* op. cit. (passage not found in B.'s works).

[**] Marshall Hall, Diseases and Derangements of the Nervous System, p. 99. Lond., 1841.

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* J. Müller, Manuel de physiologie (ed. Littré), t. i. p. 278. Paris, 1851.

† The Physiology of Man, vol. i. p. 395. New York, 1866.

‡ Principles of Human Physiology, p. 333. Seventh edition. Lond., 1869.

§ Albertus Haller, Elementa physiologiæ, loco cit.

‖ Morgagni, De sedibus et causis morb. English ed., by Benjamin Alexander, M.D. Vol. i. p. 341, Letter XIV. art. 26, 27. London, 1769.

The cause of physiological sneezing usually consists in an irritation of the nasal branches of the trifacial nerve by congestion, dust, odors, mechanical or chemical contacts. In many persons the spasm is easily brought about by the action of light upon the conjunctivæ (optic nerve also?). It has been stated that the application of a spirituous liquor to the anterior part of the palate may act as an excitant.[*] I have been informed by a well-known medical man that, when a boy at college, he was in the habit of exciting sneezing in his own person by scratching a certain spot upon his head, near the vertex, and a little to one side of the median line; thereby causing frequent disturbances in the class-room. Sneezing is spoken of by older writers as preceding attacks of epilepsy,[†] and one author [‡] states that it is very commonly observed just before sexual congress. Romberg [§] states that one of the listeners to his lectures had informed him that he was obliged to sneeze whenever a salacious thought suggested itself to his mind.

2d. Under what circumstances can sneezing itself merit the name of a disease; when does the act become pathological? The older writers on medicine appear to have had their attention directed to this matter. Sauvages,[‖] who recognizes six varieties of sternutation, says that epidemic sternutation had before his day proved so violent as to cause death. He does not give any reference to his authority for this statement, and I have been at some trouble to trace the report, and have found it attributed to an author named Polydorus Virgilius,[¶] whose work I have been unable to procure. Morgagni,[**] Bonetus,[††] Albrecht,[‡‡] Fabricius Hildanus,[§§] and Lancisi,[‖‖] are quoted as reciting fatal cases. Morgagni's case is a good one; an autopsy yielding negative results having been made. Bonetus does not speak from personal experience, but relies on the statement of a supposed eye-witness, Famianus Strada; the subject sneezed twenty-four times, and expired during the twenty-fifth sternutation, it is supposed, from rupture of the vessels of the brain. Albrecht's case is circumstantially related, and may be accepted. On reading Lancisi's work I found that he referred to the same case as Bonetus. We are, therefore, willing to admit the existence of only three fatal cases, one of these being imperfectly recorded. Hildanus's work I have not been able to consult. Haller[*] observed a case in which a deviation upward of the eyeball was produced by violent sneezing. Erasmus Darwin[†] places sternutation in his Class II. (diseases of sensation), Ordo I. (increased sensation), and Genus I. (increased action of the muscles); together with diseases which he considers allied species, such as asthma, singultus, tenesmus, parturition (!) etc. Romberg[‡] records an instance of sneezing extending over a period of four years; and the case of a woman who was seized with violent sternutation whenever conception occurred—the fit of sneezing usually taking place in the morning. Sir Benjamin Brodie[§] saw two cases of very severe and prolonged sneezing, in both of which profuse secretion of serous fluid took place from the nostrils. Among recent writers upon diseases of the nervous system Handfield Jones[‖] and Eulenberg[¶] have considered the subject in a systematic way, and the former author has advanced the view that sternutation may sometimes be due to a central cause. Dr. Peter Young[**] communicated a case of prolonged sneezing to the Obstetrical Society of Edinburgh, occurring during the middle term of pregnancy, and ultimately causing abortion. On becoming pregnant a second time the woman experienced similar fits of sneezing, beginning, as before, about the fourth month of gestation. A remarkable case has been described by Mosler,[††] as occurring in a young girl. The patient, previously rendered anæmic by various causes, became affected with an acute inflammation of the meatus and deeper parts of the ear, and shortly afterward began to sneeze in very different paroxysms. The frequently recurring spasm was very violent, and soon reduced the patient to a state of alarming prostration. For three days she was able to take but very little food, and obtained no rest; she became aphonic, the sides of the thorax corresponding with the diaphragmatic insertion became the seat of extreme pain, the pulse attained a rate of more than 120 beats per minute, the face was flushed, and the nasal and buccal mucous membranes dry. The ear was swollen and tender, and there existed much pain in the whole of the same side of the face. A warm bath, together with cold affusion to the head and spine, and two doses of .01 morphia put an end to the threatening disorder, after it had continued eighty hours, and after the patient had sneezed at the least estimate fifty thousand times. A severe case of sneezing lasting over a week, and accompanying a first menstrual period, is reported by Dr. Trautmann, Sr.[*] Cases of severe sneezing are also recorded by many of the older authors, among whom may be named Delius,[†] Lanzoni,[‡] Schubart,[§] the two Franks.[‖] In the case of Delius the sternutation was allied with hiccough in the course of a malignant fever. Finally Dr. Brown-Séquard has told me of a case observed by him in which violent sneezing occurred during the progress of an inflammatory affection of one-half of the medulla oblongata, diagnosed during life, and verified by a post-mortem examination. In this patient, the pain caused by the spasm was about the base of the thorax, on both sides.

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[*] De Lens, cited by Rullier, Dict. des sciences médicales, t. lii. p. 578, 1821.

[†] Stalpart van der Wiel, Obs. rarior. med.—anat.—chir. Lugd., Batav., 1687.

[‡] Amatus Lusitanus, cited by van der Wiel, op. cit.

[§] Romberg, Nervous Diseases of Man, vol. i. p. 349, Syd. Soc. Trans. Lond., 1853.

[‖] Sauvages, Nosologie médicale, t. ii. pp. 50, 52. Paris, 1771.

[¶] Polydorus Virgilius, de invent. rerum. Lib. 6, cap. 2.

[**] Morgagni, op. cit. Letter XIV. art. 27.

[††] Bonetus, Sepulchretum anat. vol. i. lib. 1. sect. xx. obs. xvii. Genevæ, 1700. 449.

[‡‡] Albrecht, *in* Ephem. naturæ curios. Dec. II. obs. xii. p. 38, 1687.

[§§] Fabricius Hildanus. Opera omnia, 1646. Cont. I. obs. 24, p. 26.

[‖‖] Lancisi, De subitaneis mortibus, p. 45. Romæ, 1709.

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[*] Haller, op. cit.

[†] Darwin, Zoonomia, vol. ii. p. 222, Am. ed. Phil., 1797.

[‡] Loco cit.

[§] Works, Vol. iii. p. 173. London, 1865.

[‖] C. Handfield Jones. Studies on Functional Nervous Disorders, pp. 644–46. Lond., 1870. 2nd ed.

[¶] A. Eulenberg. Lehrbuch der functionellen Nervenkrankheiten, pp. 672, 3. Berlin, 1871.

[**] Proceedings of Edinburgh Obstetrical Soc. *in* Edinb. Medical Journal. Nov., 1861, p. 492.

[††] Mosler, Virchow's Archiv. Bd. xiv. s. 557, 565. 1858.

The causes of pathological sneezing may be classified into local (nasal), central, and peripheral. The local cause may be an inflammation of the mucous membrane of the nose, vibriones irritating the mucous membrane (in hay-fever, Helmholtz[¶]), the presence of a foreign body in the nostrils (snuff, various other sternutatories, the larvæ of insects,[*] etc.). Concerning the causes of sneezing produced by morbid states of the nervous centres, we really know nothing. Perhaps many of the hysterical cases may be due to this cause. The causes acting from a distance are numerous. Uterine,[†] intestinal,[‡] pulmonary (asthma and whooping-cough), conjunctival or retinal irritation may produce the spasm.

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[*] Schmidt's Jahrb. Bd. viii. p. 50, *from* Summarium, Bd. x. Hft. 3, p. 378.

[†] Delius, *in* Acta Phys.—Med. Acad. Cæsareæ-Leopold-Carol. Vol. viii. obs. cviii. p. 380, 1748.

[‡] Lanzoni, *in* idem. Vol. i. obs. lxiii. p. 117, 1727.

[§] Schubart, *in* Ephem. naturæ curios. Dec. 1. obs. cxxxviii. p. 211, 1672.

[‖] J. Frank, Praxeos medicæ universæ præcepta. Vol. ii. p. 831. Cited by Romberg, op. cit.

[¶] Cited by Eulenberg, op. cit. p. 672.

In one of Romberg's cases [§] the third branch of the trigeminus was found diseased before its exit from the skull. In Mosler's [‖] extraordinary case, inflammation of the ear, causing irritation of branches of the trigeminus, was undoubtedly the cause of the spasm. Mosler attempted to produce sneezing in dogs by direct irritation of the auricular filaments of the fifth pair, but without success.

Treatment the most varied and extraordinary has been tried for the relief of morbid sneezing. Bartholinus, quoted by Haller, seems to have been the first to suggest irritation of a sensitive nerve of the face, between the angle of the eye and the nose, as a means of arresting the spasm. Haller speaks of the plan as if he had tried it himself. Morbid sternutation has been cured by the use of snuff,[¶] and Darwin [**] asserts that, "when it is exerted to excess it may be cured by snuffing starch up the nostrils." Dr. Gairdner [††] strongly advocated the use of blisters to the nape of the neck, not only for the arrest of the spasm under consideration, but also for the cure of convulsive cough and hiccough. This means is perfectly analogous to that proposed by Bartholinus, Haller, Diday, and the author: an irritation varying in intensity being transmitted to the centres in the spinal axis which are about to furnish the motor impulse for the spasm, the activity of these motor centres is inhibited or arrested by the new (more intense?) irritation. Romberg [‡‡] advises the use of emetics. Eulenberg[*] recommends emetics, skin irritation (mustard), such tonics as iron, arsenic and quinia. An English bishop, subject to very harassing fits of sneezing, is mentioned by Watson[†] as having found an effectual remedy in dipping his head into cold water. Mosler controlled the case which he reports only by a combination of the warm body bath, and cold affusion to the head and back of neck; a means which produced syncope in his weakened patient.

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[*] Carpi *in* Harless Jahrbücher, Bd. i. Hft. i. Cited by J. Frank, Praxeos Medicæ Universæ precepta, vol. ii. pars i. p. 965.

[†] Dr. Peter Young, loco cit.

[‡] Dr. Little, in Obstetrical Soc. of Edinburgh, Edinb. Med. Journal, Nov., 1861, p. 493.

[§] Loco cit. p. 347.

[‖] Loco cit.

[¶] Bauwens, cited *in* British and Foreign Med. Review, 1836, vol. ii. p. 245.

[**] Darwin, op. cit.

[††] J. Gairdner, on Anomalous Affections of the Respiratory Organs. Edinb. Med. and Surg. J., ii. p. 77. 1840.

[‡‡] Romberg, op. cit.

A number of authors insist upon the necessity of removing the peripheral cause, irritation of uterus, etc., if any exist. It would seem as if these remedial measures might be properly classed under four heads. In the first place, means tending to allay the irritation of the nostrils (starch); in the second place, remedies which produce, or tend to produce, syncope, and its attendant lowering of nervous irritability (emetics, warm baths, narcotics); in the third place, medicines which, like iron, quinia and arsenic, diminish morbid irritability by improving the nutrition of the nervous centres; and fourthly, inhibitory means, such as cold to the head and neck, mustard and cantharidal irritation to the neck, inhalation of iodine (Eulenberg), and pressure upon the branches of distribution of the infra-maxillary nerve. This last measure is the one which has surely arrested or prevented physiological sneezing in my experience, and in that of many persons to whom I have recommended it. The upper lip is very convenient for this purpose, and the moderately painful impression required is easily excited there by the pressure of the side of the index-finger. Whether this simple means would succeed in cases of pathological sternutation I will not venture to predict. Should it fail, I would suggest as producing a more intense irritation of the same nerves, the use of faradization of the skin of the same parts (lips and cheeks near nose). The wire brush electrode should be used in connection with the secondary current, the skin to be thoroughly dried, and even covered with starch-powder. It seems to me rational to anticipate good results, also, from the faradization of the nape of the neck in morbid sneezing, hiccough, or convulsive-cough, after any existing peripheral cause shall have been removed. These irritations of sensitive nerves (pressure, faradic-current) transmitted centripetally by the filaments of the second branch of the fifth pair, or by those of the upper cervical nerves, exert an inhibitory influence upon the motor centres which are about to become active, viz., the spinal centre of the phrenic nerves in the first place, and, secondly, the extensive kinetic tract which is connected with the muscles of expiration.

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[*] Eulenberg, op. cit. p. 673.

[†] Watson, Lectures on Physic. Am. ed., p. 110. Phil., 1858.

Various therapeutical applications of this method suggested themselves to me at the time.

a. Certain applications to medicine. In various forms of internal hemorrhage, especially that occurring in the nasal and pulmonary tract, it is highly desirable to avoid the jar produced by sneezing. The same indication exists in the course of development of those aneurisms which fall to the care of the physician; for some days after the reduction of prolapsed rectum or uterus; or in the advanced stage of hepatic abscess, or hydatid disease. A minor use to which this inhibitory action may be put, is the prevention of such pains as are produced by shaking of the body, or by deep inspiration.

b. Certain applications to surgery. Sneezing must be avoided after many plastic operations, and operations upon the vagina, uterus, or abdominal walls. After many cutting operations about the eye, absolute rest is highly desirable; and is even more required in the cure of cleft-palate. Of course, under many circumstances, the arrest of hemorrhage may be made surer by the avoidance of sternutation.

The reader may, perhaps, pardon me if I add a non-medical paragraph. The custom of invoking a blessing upon persons who sneeze is a most interesting one. Several of the old medical authors above referred to state that the custom dates from the time of a severe epidemic (in which sneezing was a bad sign) during the Pontificate of Gregory the Great. Brand,* however, and the author of an article in Rees's Cyclopædia,† states that the phrase "God bless you," as addressed to persons having sneezed, is much more ancient, being old in the days of Aristotle. The Greeks appear to have traced it back to the mythical days of Prometheus, who is reported to have blessed his man of clay when he sneezed.‡ In Brand, the rabbinical account of the origin of the phrase is given as originating in the alleged fact that it was only through Jacob's struggle with the Angel that sneezing ceased to be an act fatal to man. In many countries sneezing has been the subject of congratulations, and of hopeful augury. In Mesopotamia and some African towns, the populace are reported to have shouted when their monarchs sneezed.[*]

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* Brand; Observations on Popular Antiquities, vol. iii. pp. 119–127. Lond., 1849.

† Rees's Cyclopædia, vol. xxxiv. art. Sneezing.

‡ Alex. Ross's Appendix to Arcana Microcosmi, cited by Brand.

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[*] It would be unjust not to state that Diday (op. cit.) had suggested practical applications of his method, and that I became aware of this fact only after having matured the original parts of this article.

Rullier. Article Sternutation, Dict. des Sciences méd. t. lii. p. 577, 1821.
Merlet. Est ne sternutatio naturalis actio? Paris, 1654.
Schneider. De osse cribriformi, etc. Wittemb., 1656.
Schoock. De sternutatione. Amst., 1664.
Alberti. Diss. de sternutatione. Lips., 1671.
Birnbaum. Diss. de sternutatione. Lips., 1672.
Forestus. Diss. de sternutatione. Argent., 1688.
Hoffmann. (Maurice.) Diss. ptarmographia physiologico-pathologico-therapeutica. Alt., 1710.
Eyselius. Diss. de stern. præternaturali. Erf., 1716.
Rhanius. Diss. de more sternutantibus salutem apprecandi ejusque origine. Tigur, 1742.
Unzer. Diss. de sternut. Hal., 1748.
Porta. Diss. de sternut. Basil, 1755.
Buchner (A. L.) De sternut. commodis et incommodis. Hal., 1757.
Faselius. Diss. de causis sternutationis ejusque effectibus. Ienæ, 1765.
Sidren. Diss. de sternut. Upsal., 1779.
Van Leempœl. Diss. de sternut. Lovan., 1788.
Metzger. Diss. de sternutatione. Regiom., 1796.
Rega H. J. De sympathia, p. 240. Harlemi, 1721.

DESCRIPTION OF A PECULIAR PARAPLEGIFORM AFFECTION. (TETANOID PARAPLEGIA.)*

THE condition to which I desire to call attention is one which cannot be of very rare occurrence, as, in the course of three years, I have met with five examples of it. No doubt many readers will at once remember having observed precisely similar symptoms.

This form of false paraplegia (using this word as implying the existence of paresis or akinesis in the lower limbs), is characterized by impairment of the functions of the lower extremities, when the patient is in the erect posture, without any loss of power in these parts. Further analysis shows that the seeming paraplegia is dependent upon tonic spasm of the muscles of the lower limbs. As negative characters we have absence of the symptom ataxia, and often, also, preservation of sensibility.

The clinical aspects of a case of tetanoid paraplegia are the following: The patient complains of having nearly lost the use of his lower limbs (he may speak of great "loss of power"); of having various abnormal sensations in them, and of experiencing trouble in the evacuation of his fæces and urine. When the patient is told to get up and walk, he rises with difficulty from the bed or chair, assisting himself with his hands. On getting into a perpendicular position, with or without the aid of a stick, he oscillates a good deal, and seeks to re-establish his equilibrium by separating his feet and bending his body forward. In this posture the knees remain extended, and the feet are not averted as in health; they are often, on the contrary, turned inward. The attempted steps are peculiar. The feet are not dragged along as in ordinary cases of incomplete paraplegia, nor are the knees much flexed, and the feet brought down violently as in locomotor ataxia. There is none of the outward projection of the entire limb, so characteristic of the latter disease. The limbs remain extended, and the feet are simply pushed along the floor; the slight raising of the soles from the support being accomplished by a movement involving the entire lower extremity. The tendency of the great toe is downward and inward, thus producing, or tending to produce, a partial crossing of the limbs, and tripping. If during the efforts the observer feel the patient's muscles, he will find them firmly contracted.

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* From the Archives of Scientific and Practical Medicine, Feb., 1873.

If the patient be seated, or made to lie upon a bed, and the strength of his lower limbs tested in the usual way, *i.e.*, by bidding him resist attempts at passive flexion or extension of certain articulations, the muscular power will be found almost up to the normal limit, if not quite so, in every part of the lower half of the body. There is not, necessarily, any weakness of the abdominal muscles. There is, consequently, no paresis present, and we cannot correctly speak of the case as one of paralysis. The reflex power of the lower limbs, tested in these two positions, is found to be much exaggerated, and a state approaching spinal epilepsy (a mixture of clonic and tonic spasms) may be developed by the examination. Caloric appears to excite these reflex actions most readily.

The state of the bladder and rectum is sometimes peculiar (Cases III. and IV.).

The urine does not dribble away, and exhibits no pathological alkalinity. If passed involuntarily, it is at intervals, and by jets; a normal desire to urinate occurring. Usually the patient is simply obliged to hurry the evacuation of the viscus, or an emission of urine by reflex action will take place very quickly after the sensation of fullness has been perceived. One of the patients (Case IV.) expressed the state of affairs very well by saying, "Now that I am better, I can go the length of the ward before letting go, but no further." The bowels are, as a rule, constipated, but when fæces descend into the rectum, a rapid emptying of the organ is inevitable. It appears to me reasonable to suppose that this morbid excitability of the rectum and bladder existed in all the cases at some period or other. Retention of urine—a truly paretic trouble—is apt to develop as the case progresses.

As above stated, sensibility is not necessarily impaired, and in only one case (Case I.) was there much anæsthesia. In two cases (Cases II. and III.) sensibility remained normal. It is also noteworthy that the existence of considerable cutaneous anæsthesia (Case I.) did not at all interfere with the production of reflex movements.

In no case could true ataxia be made out. The muscular sense appeared normal, and what inco-ordination existed was due to spasmodic action of the abductor muscles. It will be remembered that in ataxia (the symptom) affecting the lower extremities, exaggerated action of the abductor muscles is present.

The pathological condition to which these symptoms seemed, in all the cases, allied, appeared to be compression of the anterior part of the spinal cord in the dorsal or cervical region. In three instances (Cases I., II., and V.) this can hardly be questioned, since kyphosis existed. In Cases III. and IV., I admit that the diagnosis of tumor is not established in the most conclusive way, but as these patients are still under my observation, I may at some future time be able to clear up the uncertainty. As things now stand, I believe that I am authorized to conclude that the peculiar false paraplegia caused by reflex movements, and to which I venture to give the name of tetanoid paraplegia, is to be looked upon as a symptom of moderate compression of the spinal cord at some point above the lumbar enlargement. I do not wish to be understood as denying that tetanoid paraplegia may occur in cases of functional disturbance of the spinal axis. Although I have seen no such case recorded in the publications accessible to me, I have no doubt that a similar or analogous state may be observed in hysteria. It appears probable to me, however, that in cases of increased spinal excitability, without lesion, the spasms would be more clonic (saltatory) than tonic (tetanoid) in character.

The pathogeny of this symptom is similar to that of the closely allied (often coexistent) group of symptoms called spinal epilepsy. In the first place, as a cause of increased reflex action of the spinal cord must be reckoned the diminution of the cerebral influence brought about by compression of the cord. This is in accordance with what experimentation upon the lower animals teaches us. It is a pretty generally admitted view that the cerebral influence moderates the motricity of the spinal cord. Another element in the production of these reflex spasms I believe (with Dr. Brown-Séquard,* when he speaks of spinal epilepsy) to be congestion of the spinal cord below the lesion.

This symptomatic group evidently belongs to the class of hyperkineses (Romberg), and therefore it may be interesting to determine its relationship with some other varieties of increased muscular action.

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* Lectures on the Diagnosis and Treatment of Paralysis of the Lower Extremities. Phila., 1861, p. 60.

Its most closely allied congener is spinal epilepsy. This appellation was given by Dr. Brown-Séquard to a combination of tonic and clonic spasms affecting paralyzed parts in certain affections of the spinal cord. He describes it in these terms: "Whether spontaneously, or after an external irritation (such as a shock, or a pressure on some muscles, tickling the sole of the foot, or the passing of a catheter into the urethra), the lower limbs are often moved violently or become perfectly stiff; sometimes they are drawn up forcibly in a state of flexion, the back part of the foot pressing against the hip-joint; sometimes the thighs are drawn violently one against the other by a spasm of the adductor muscles, and they press very hard against the testicles; in other cases the flexor and extensor muscles contract alternately with great violence, and, after a few minutes of great shaking, a rigid condition appears, which, after a time, is followed by relaxation and quietness."* Dr. Brown-Séquard believes that "this spasmodic affection of the paralyzed legs is the result of the morbid increase in the vital properties of the dorso-lumbar enlargement of the spinal cord, owing to two causes: 1st, the congestion of that part of the cord; 2d, the accumulation of power in that part of the cord, in consequence of its not being any more under the action of the will." He states that the pathological conditions which, according to their localities, may produce this symptom in man, are localized myelitis, tumors pressing upon the cord, fracture and dislocation of the vertebræ, and that by section of the spinal cord he can easily produce this condition in animals. The same group of symptoms, a combination of tonic and clonic spasms occurring under similar circumstances, had many years before attracted the attention of several observers, and Dr. William Budd has left on record an admirable description of these spasms as observed by him prior to 1839.† Jaccoud‡ considers exaggerated reflex power in paralyzed limbs in general, and the form of hyperkineses now under consideration (spinal epilepsy) in particular, as a positive sign of organic paraplegia.

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* Brown-Séquard, op. cit., p. 59.

† Medico-Chirurgical Transactions. Vol. xxii. p. 153. 1839.

‡ Des paraplégies et de l'ataxie du mouvement. Paris, 1864, pp. 484, 488.

A noteworthy variety of spinal epilepsy consists in a succession of clonic spasms (trembling) of limited range affecting the paretic or akinetic limbs. In case of partial, insulated cerebro-spinal sclerosis (and other morbid conditions of the spinal cord), this trembling may be excited by irritation of peripheral nerves; and forcibly flexing the foot (patient being in recumbent position) seems to have special efficacy. A similar movement may be produced in healthy individuals by insufficient and ill-placed support of an extremity: a foot, for example.

The spasm which constitutes the most important element in the obscure affection known as writer's cramp is analogous to tetanoid paraplegia. Here the patient, while writing the first few words experiences no marked difficulty, but after the spinal cord has been acting for some time, a spasm, more or less tonic in character, affects the flexor muscles moving the thumb and fingers, and there ensues an illegibility in the writing, or an utter impossibility to hold the pen. The same spasm of the flexors occurs when the patient is using his fingers for other purposes, such as holding a cup or saucer; never spontaneously. We thus have hyperkinesis determined by the action of a peripheral irritation upon a functionally diseased nervous centre.

There is, it seems to me unquestionable, a spasmodic element in the complex group of symptoms which constitutes the disease known as locomotor ataxia. In the first place, as believed by Brown-Séquard,* the conservation of force in the lower limbs, in the paraplegic forms of this disease, is only apparent. I am ready to admit, with him, that a certain degree of paresis exists in the affected parts, but that the methods employed for testing the degree of volitional force really develops a degree of reflex (morbid) power which, after the first moments of the examination, conceals whatever loss of power may previously have existed, and causes even an abnormal degree of muscular strength. Secondly, in the symptom ataxia, I have for some time believed that the characteristic disharmony in the action of various muscular groups in the typical stages of the disease locomotor ataxia, was due not so much to diminished nervous influence in the muscles overcome by their antagonists, but in increased motricity sent to the over-acting muscles. Thus, in the jerking, externally projected steps there is an overbearing action of the abductors and extensors, and this is due, in my opinion, to the reception of abnormal increased motricity by these muscles; a motricity developed in a reflex way by the exercise of the limbs (see above, writer's cramp). I am aware that recent writers are inclined to consider the disharmonious action above referred to as wholly dependent upon varying degrees of impairment of the muscular sense. If this be so, why should the disturbance affect definite muscular groups?

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* Oral communication, 1869.

Certain forms of contracture of the paralyzed limbs in hemiplegia (of cerebral origin) bear a very close resemblance to the false paraplegia I have endeavored to describe. There is now a male patient under my care at the Epileptic and Paralytic Hospital, Blackwell's Island, who experienced months ago an ordinary apoplectic stroke, followed by right-sided hemiplegia (including face), and temporary aphasia. He has recovered some degree of voluntary motion in the palsied limbs, but suffers much from contracture of the arm and hand. The resident physician, Dr. Bruce, the nurses, and the patient himself assure me that at night and in the early morning, before exposing the parts to the air, or attempting to rise, the hand lies quite open and relaxed, and that no stiffness whatever exists at the elbow-joint. The contact of air, however, and, more surely, the acts of rising and stepping upon the cold floor, provoke a spasm which in a few minutes reaches its maximum. When this is at its highest degree of tension (as it is during my visits) the forearm lies across the chest (patient sitting in a chair or walking), the elbow being bent about at right angles; the wrist is somewhat flexed, and the finger-nails are forced into the palms of the hand. The observer's efforts to overcome this contracture only increase it, and the same is true of the patient's own volitional efforts, and of his using the other limbs. The right lower extremity is moderately stiffened, in extension, during waking hours. The flexors and adductors of the upper extremity have not suffered in nutrition, while the overpowered (stretched) extensors are in a state of unmistakable atrophy, and have lost electro-muscular contractility. The pathological physiology in this case I believe to be precisely similar to that explained when speaking of tetanoid paraplegia. The principal cause of spasm (increased reflex power) in both cases is the separation of the spinal axis from the cerebrum: in the false paraplegia the cutting off occurs somewhere in the spinal cord, while in hemiplegia it happens at the junction of the upper end of the spinal axis with the cerebrum (corpus striatum).

I wish to add a few words concerning a form of "stiffness" of the lower limbs which is much complained of by patients having congestion (?) of the spinal cord and its meninges. This is a purely subjective sensation which accompanies the numbness and formication which form such prominent features in these cases. The "stiffness" as well as the numbness are worse when the subject is lying down or sitting, and are greatly felt during the first efforts at movement. Contrary to what obtains in spasmodic paraplegia, this feeling grows less marked after the patient has taken active or passive exercise. There is no real (objective) rigidity, and reflex movements are not necessarily modified from the healthy standard.

As regards bibliography I can say but little. Only one writer, to my knowledge, seems to have noticed and described a condition similar to the one forming the subject of this contribution. I refer to Jaccoud,* who, in his valuable work on various forms of paraplegia, gives a page and a half to what he calls false paraplegia, due to exaggerated spinal excitability. He does not refer to any cases, nor does he give any account of the pathological conditions accompanying the symptom. He undoubtedly has seen cases similar to mine. I have come across a reference which may be thought to indicate that the physician referred to had seen and described spasmodic false paraplegia. E. Goupil† made (ever published ?) a classification of hysterical paraplegias, embracing the following varieties: 1st. Hysterical paraplegia due simply to muscular weakness; 2d. Hysterical paraplegia produced by the extreme pain caused by reflex action and movement; 3d. Hysterical paraplegia produced by loss of muscular sensibility. While admitting that Goupil's second variety bears a certain resemblance to tetanoid pseudo-paraplegia, I would recall that in the cases I am about to detail, pain on movement was not a feature, and that hysteria had nothing to do with any one of my five instances. The older and more recent treatises and monographs upon diseases of the nervous system, with the above exception, contain no reference to the condition I have described.‡

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* Jaccoud, op. cit. pp. 469–471.

† Cited by Leroy: Des paralysies des membres inférieures. Paris, 1856; p. 210.

‡ After the above had been printed I had the opportunity of reading Hallopeau's interesting thesis entitled *Des accidents convulsifs dans les maladies de la*

CASE I.—By the kindness of Dr. Gustavus A. Sabine, I had the opportunity of studying the symptoms presented by Mr. P., an Englishman, aged 46 years. About six weeks before the consultation (April 6, 1872) patient noticed, while at first walking in the morning, a slight degree of numbness and formication with "loss of power" in the lower extremities. The numbness affected all the parts below the knees, and to a much less degree the anterior surface of thighs. At same time, or shortly after, he observed twitching of the lower limbs at night. These three symptoms—formication, "loss of power," and twitching—progressively increased, until he now walks with difficulty, even when aided by a stick. Yesterday was obliged to ask assistance to cross a busy thoroughfare. The bowels have been costive and the urine hard to pass. Since three weeks has felt as if a band were tightly drawn around the lower part of the abdomen. Lower limbs have moderately wasted. The "loss of power" has rapidly increased in the last three days.

The examination shows nothing abnormal about the upper part of body excepting the fact that the pupils are extremely minute.* The co-ordination of the upper extremities is perfect. The patient walks in a peculiar way. He leans firmly upon a stick, and takes very short steps with limbs almost perfectly rigid. There is neither distinct jerking outward of the feet nor dragging; but patient staggers much. Stands with feet somewhat separated, and without stick oscillates a good deal. The walk is not made different or worse by closure of eyes. Strength of lower limbs, tested in sitting and recumbent postures, shows very slight, if any, impairment. Movements well co-ordinated. Some disturbance of sensibility; superficial contact not normally perceived, impressions of pain retarded and metamorphosed into burning. Localizes impressions well, and sense of temperature is normal. Reflex movements exaggerated. Lower abdominal muscles weak; cause of constipation and slow micturition. No spontaneous pain in back or limbs. Deep pressure reveals obscure tenderness on level of fifth and sixth dorsal vertebræ. Patient states that stiffness in limbs and back is worst in early morning, and is somewhat relieved by exertion. Twitching of legs increased; often has alternations of clonic and tonic spasms (spinal epilepsy). On the 23d a second examination shows continuance of numbness and stiffness. Cramps decidedly less. Rather more impairment of sensibility; pinching produces severe burning. Patient loses his limbs in bed; once attempted to rise in dark, and failed to "strike bottom" with his feet; found himself on his knees. Is unconscious (eyes closed) of passive movements below hips. Co-ordinates well. May 3d. The obscurity of the case is to-day cleared up by the discovery of a slight but distinct angular curvature of the spine, caused by projection of spinous processes of fifth and sixth dorsal vertebræ. Deep pressure produces pain. A few days later the patient started for England.

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moelle épinière, Paris, 1871, in which I find a case of false paraplegia caused by spasm, well described (Case III., p. 56). The author correctly appreciates the significance of the symptom, but has not called attention to it specially in his remarks.

* It may be interesting to state that the patient's brother, an apparently healthy man, has similarly small pupils.

CASE II.—J. H., male, aged 32 years, in my service at the Epileptic and Paralytic Hospital, Blackwell's Island. Patient was first admitted in March, 1869, discharged, and readmitted in July, 1870. Two histories of the case are on record, which differ somewhat as to the mode of reception of the injuries, which produced various symptoms. There is no conflict, however, on the points which render the case interesting in the present connection, viz., on the state of the spinal column and of the lower limbs. I will abridge from both accounts, one of which was written at the bedside under my own dictation.

Eighteen years ago a chimney fell upon patient and produced a compound fracture of the skull, necessitating the removal of large pieces of bone. No paralysis resulted from wound or operation. Now bears a large scar on left side of vertex, six inches in length (antero-posterior), three-quarters inch at widest part, and in some places it is three or four lines deep. The scar extends a little across the sagittal suture. The pulsations of the brain are distinctly felt through the cicatrix. About two years ago (1867), a carriage passing over him, he was kicked in the back, and his spine "broken." After this accident he was able to walk, though with great difficulty. In January, 1870, felt "rheumatic pains all over," and in the spring went into Bellevue Hospital. The legs felt "dead" to him, and no jerking was present. While in Bellevue had more or less retention of urine, and the catheter was sometimes used. The right leg was flexed for five weeks, the left never, but both were stiff and strongly adducted.

Condition on July 7th, 1871. Lies in bed; can raise each heel four or five inches from bed, and can voluntarily move every articulation of lower limbs. Strength seems to be perfect in lower extremities; voluntary adduction being almost impossible to overcome. Reflex movements are very violent. In the erect position the movements of the legs are but little subordinate to the will. The patient can hardly stand, even with the support of crutches; when movement is attempted, but slight motion is seen at knee-joint. [I may here add that I remember most vividly what is not sufficiently entered in the record, viz., the patient's very peculiar attempt at walking. He would get out of the bed with help, his legs being moderately rigid, but the moment his bare feet touched the floor, most severe reflex movements occurred, producing tetanic rigidity of the limbs. Holding on by the head of the bed, a chair, or an attendant's arm, he could take a few steps, which consisted in sliding of the feet a little way, no hip or knee or ankle movement being apparent. He was also conscious of a tendency to adduction and crossing of legs.] Sensibility seems in all respect to be good (increased?) in lower limbs, and patient feels some numbness, more on right side. Upper extremities in normal state; bears marks of bed-sores on hips and sacrum. Spine exhibits kyphosis in its middle dorsal region, accompanied by slight scoliosis to the left. The greatest angular curvature is on level of fifth and sixth dorsal vertebræ. No difficulty in making water; bowels costive. At a later period the patient died of extensive erysipelas, but no autopsy could be obtained of the friends. I had diagnosed compression of the spinal cord by the products of broken-down vertebræ, there having been a traumatic Pott's disease. At one time there must have been much localized meningitis and perhaps superficial myelitis.

[illegible] admitted to the Epileptic and Para[illegible] at first a matter, but during the last few [illegible] constitutional syphilis, rheumatism, or [illegible] voyage from Italy to New York. [illegible] was rheumatic, caused by constant [illegible] and lower extremities. [illegible] referred to all parts below the waist [illegible] has since had much pain, principally in left [illegible] lower limbs. In March, 1872, he was [illegible] upper extremity, accompanied by a [illegible] progressively lost the power of moving [illegible] similar symptoms (trembling and [illegible] affected the right arm. At the same [illegible] a little outside the [illegible] symptoms excepting occasional [illegible] patient began to experience [illegible] away, but after a small [illegible] desire to urinate was irresistible [illegible] involuntarily emptied. At the begin[illegible] examination of this patient. The mouth [illegible] tongue projects straight out; [illegible] so that whistling is very im[illegible] a little thick. Pupils normal. [illegible] face. The upper extremities [illegible] very marked atrophy of many [illegible] of the thenar and hypothenar eminences, and nearly [illegible] movements are distinct in many muscles of the [illegible] sensibility in its various modes is perfect. There is [illegible] about trunk. The lower extremities present [illegible] The patient cannot stand erect with his bare feet upon the floor [illegible] can stand with help of nurse, and by great effort. During the attempt he oscillates greatly. While walking with help, slides both feet along in an equally awkward way; does not jerk or drag them. During the efforts, standing or walking, the muscles are rigid. Patient says that his legs feel stiff. Closing eyes does not make the attempt at standing worse. Examined in the recumbent posture, all movements are performed by lower limbs with effort, but with perfect co-ordination. Efforts cause more or less clonic spasms in lower limbs, and a pain in masses of muscle forming anterior part of thighs. Scratching soles, or other modes of irritation (cold impressions especially) produce strong reflex movements, mostly clonic. Strength of various joints perfect. Fibrillary movements in muscle are excited by flipping the skin. The various modes of sensibility are normal. The patient has observed that his legs stiffen when the cold air first strikes them on getting out of bed. The bladder trouble is as above described, an incontinence through spasm. The diagnosis of tumor compressing the spinal cord (incipient Pott's disease?) is made from co-existence of referred pain to legs and left side, of false paraplegia (tetanoid), and atrophy of a few muscles of upper extremity. This last symptom is explicable by compression of some anterior roots of nerves. The seat of compression is probably at the upper part of the cervical enlargement.

CASE IV.—J. R., aged 42 years, a baker of intemperate habits, and admitting great sexual excesses, in 1864 had primary sore and secondary syphilitic symptoms. During the fall of 1869 he suffered from pain in the right side, and later, in the back ; this being made worse by motion. March 6th, 1870, he awoke with both legs numb, accompanied by retention of urine. Admitted into the Epileptic and Paralytic Hospital, bearing a large bed-sore on sacrum, and having a paraplegia characterized by numbness and excessive reflex action. Before admission reports that he could not move lower limbs in the least. Improvement began in July ; noticed sensation of distended bladder, acquired some voluntary movement of both legs ; more control over left. Was unable to control rectum and bladder. Improved much under hypodermic injections of strychnia ; bed-sore healing. During 1871–2 had iodide of potassium in large doses. I was inclined to consider the paraplegia one dependent upon a syphilitic lesion of the spinal dura mater, causing pressure and irritation. Discharged in early summer of 1872, able to walk with crutches, and gaining. Examination in supine position shows that voluntary movements of left lower extremity are very free ; can raise foot more than twenty inches from the bed. Right foot can be raised only about ten inches. Strength at various joints (resistance to passive movements) normal. Has much twitching and spasm in lower limbs. Appreciates surface contact and tickling, but does not localize impressions correctly. Sense of temperature and of pain normal. Reflex movements produced by examination of sensibility. Both legs feel numb below knees. Patient bears large node on the right tibia.

Re-admitted in fall of 1872. Examination January 5th, 1873. Walks with help of crutches, or of a stick. Steps small, legs tend to cross one another (adduction), and the lower extremities are apparently stiff. Patient has noticed great spasm in them at times, on standing up. In supine position voluntary movements of the left lower limb are normal in extent ; flexion of thigh and knee on right side is limited by stiffness at knee and hip joints. Power of resistance at both knee-joints normal ; as also at other joints of lower limbs. Reflex excitability increased, more on the left than on right side. Co-ordination of movements (eyes closed) quite perfect. Is conscious of passive movements at knee-joints ; unconscious of them when made at ankles and toes. Sensibility preserved except as regards the tactile sense, which is much impaired on feet.

The patient states that his bad walking is due to stiffness of the limbs. When he attempts to stand alone in bare feet, the reflex spasm is so great as to cause him to lose his balance ; with help, and in stockings and shoes, can walk as above described. Has better control over his legs some days than others. His urine does not dribble away nor is it retained, but when the desire to urinate is felt he must empty the bladder almost immediately, or the urine is forced out against his will. Is improving while taking large doses of iodide of potassium.

CASE V.—J. K., male, 36, admitted to the Roosevelt Hospital, service of Dr. W. H. Draper, February 9, 1872. In March, 1871, patient began to suffer from pain in his spine, about the level of the third and fourth dorsal vertebræ; and about the middle of April he noticed a tumor, about as large as a small hen's egg, in the same locality. The tumor grew and the pain became more severe until May, when the application of a plaster is said to have caused both pain and swelling to disappear.

Late in December, the pain and tumor coincidentally returned, and about one month ago patient observed weakness in his legs, most marked in the right. The tumor has been stationary since the beginning of the year. Examination of upper limbs reveals nothing abnormal. Over third dorsal vertebra is a firm, slightly reddened tumor, about the size of half an egg; this tumor is painful when firmly pressed. Intercostal muscles act very slightly; respiration mostly abdominal; marked impairment of sensation, motion and co-ordination in the lower limbs; worse on right side; bowels torpid. When he wishes to urinate, he is obliged to do so at once. February 17, is much troubled by reflex contractions. Examined by Dr. Seguin at Dr. Draper's request. Marked loss of sensibility in right leg; lessened sensibility in left. Co-ordination in both legs impaired. Resistance to flexion of knee nearly normal while lying on back, but when upright this is notably lessened. I have a very clear remembrance of this examination, and will add to the short entry made at the hospital. The "in-co-ordination" consisted in extraordinary stiffness of the lower limbs, when the patient attempted to walk with the aid of a nurse. At the same time the feet tended to cross each other from strong action of adductor muscles. In the recumbent posture no trace of in-co-ordination appeared, and there was almost perfect strength in all parts of the lower extremities. It was this wonderful contrast between seeming extreme paraplegia when standing or attempting to walk, and the preservation of motor power when lying on back, that caused surprise to all present. At the same time I made out a decided angular spinal curvature involving the third and fourth dorsal vertebræ, and decided that the case was one of Pott's disease of the spine. February 20th, more pain in tumor. Incision causes escape of blood only. March 1st, retention of urine has appeared; cathether used. Angular curvature more pronounced. March 10th, reflex movements in legs very annoying; great dyspnœa. April 10th, urine dribbles away; bed-sores forming over sacrum and trochanters. Spinal angle increasing. During succeeding months the bed-sores extended greatly, the spinal curve became more acute, and power in legs was reduced to a minimum. After living for a month and a half at the point of death, patient expired July 9th.]

I am indebted for the above history to Dr. N. B. Sizer, House Physician to the Presbyterian Hospital; and wish to express my thanks to Dr. Robert F. Weir, Surgeon to the Roosevelt Hospital, for permission to make use of the case.

LECTURE UPON THE GENERAL THERAPEUTICS OF THE NERVOUS SYSTEM.*

GENTLEMEN:—I have thought that the last hour of your survey of nervous diseases could not be better spent than in a rapid review of certain principles of therapeutics, and in a partial study of the remedial agents classified as much as possible in accordance with these principles. In offering you the following classification, I wish to warn you that it has little in common with the therapeutic propositions which you will find in text-books, whether upon the practice of medicine or upon diseases of the nervous system. Inasmuch as the ground to be gone over is so large, you will pardon me if I restrict myself to short practical remarks upon the various heads of the lecture.

The following is the classification which I have long had in my mind, and which I now submit to you:

CLASSIFICATION.

- A—Agents which affect the blood-supply of nervous system.
  1. Agents which increase the blood-supply.
  2. Agents which diminish the blood-supply.
- B—Agents which affect the substance of nervous system.
  1. Agents which increase action of
     - *a*, brain.
     - *b*, spinal cord.
  2. Agents which diminish action of
     - *a*, brain.
     - *b*, spinal cord.
- C—Restoratives and Tonics.
- D—Counter-irritants.
- E—Electricity.
- F—Hygienic means.

CLASS A.

Means which Affect the Blood-supply.

The indication for affecting the blood-supply of a nerve-centre is sometimes a perfectly correct and logical one.

In practice, we have a certain class of cases in which the symptoms are due to hyperæmia of nerve-centres, and also a certain class in which the symptoms are due to anæmia of nerve-centres.

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* Delivered at the College of Physicians and Surgeons, New York, Feb. 21, 1874. From the N. Y. *Medical Record*, June 1, 1874. Vol. ix., p. 281 et seq.

[This lecture was reprinted in full by the London *Medical Record*.—R. W. A.]

The vital question is to determine whether the hyperæmia or anæmia be primary or secondary.

Few questions in pathology are of more importance than this, and few have caused such prolonged discussions. This is not the place to examine this matter, and I must limit myself to stating the conclusions to which experience has led me. I believe that true primary hyperæmia of the brain or spinal cord is a very rare affection, infinitely rarer than the teaching of books would lead us to suppose. In many so-called cases of cerebral congestion there is, I believe, no hyperæmia, but only mal-nutrition, which may be attended by a secondary and passive congestion. In all such cases the leading indication is not to deprive the nervous centre of blood. The effects of anæmia are better known and more commonly observed; and it is an instructive fact to recollect that what we to-day consider as typical of anæmia (epileptic loss of consciousness) was not many years ago looked upon as typical of hyperæmia. In brief, I believe that the indication to diminish the amount of blood circulating in the brain (and spinal cord) is very rarely presented to us practitioners, and that the contrary indication is much more commonly encountered.

The first class of means is subdivided into two sub-classes:

(1) Means which increase the blood-supply.

This sub-class may be still further subdivided as follows:

(*a*) Decubitus, or posture.
(*b*) Compression of arteries of limbs.
(*c*) Stimulants.
(*d*) Agents paralyzing the vaso-motor nerves.
(*e*) Measures which act by increasing the activity of the nervous centres.

(*a*)–The amount of blood in the spinal cord can be materially increased by placing the patient upon his back.

The amount of blood in the brain can be materially increased by elevating the legs and arms.

In syncope, therefore, and in all cases where we may suppose that anæmia of the brain is present, the first thing to be done is to place the patient upon his back, and we may, besides, elevate the limbs and trunk. Anæmic patients, who have no positive disease, but who at times feel weak and faint, or suffer from attacks of vertigo, should also have recourse to this means.

(*b*)–Compression of the arteries of the limbs is a very important and powerful means.

This should be done by tourniquets, as ordinary bands alone will not arrest the circulation carried on through the arteries, but will prevent the return of blood to the body.

This procedure is one which is rarely resorted to, and I would recommend its execution only in extreme cases of cerebral anæmia, as a means next to transfusion. The tourniquets should be applied not simultaneously, in order that you may study the reaction of the heart.

In the days when epileptic seizures were regarded as due to cerebral hyperæmia, compression of the carotids was advised and done to cut short the paroxysms. Insensibility can in this manner be produced in the healthy individual. In epileptics the results obtained were not satisfactory, and the measure is one we now condemn as unphysiological. Besides, in pressing upon the carotid we act upon the vagi, and influence the heart in that way.

(*c*)–Of the stimulants, alcohol is the best. Preparations of ammonia are employed, but they are less efficient than alcohol. The dose of alcohol in cases of anæmia of the nervous centres is to be gauged only by the degree of reaction observed. In the drowsiness and exhaustion which result from prolonged exposure to cold with or without over-exertion, in cases of loss of blood, brandy will be tolerated in enormous quantities, and the subject saved.

In general terms food is to be regarded as a stimulant, for the general circulation is made more active by taking it. Probably many of you have had an afternoon headache magically cured by dinner.

(*d*)–Paralyzing the vaso-motor nerves, by producing enlargement of the arteries, is productive of hyperæmia in the arterial district to which the nerves are distributed.

This proposition is demonstrable by experiment upon animals. If we cut the sympathetic nerve in the neck (as was done by Pourfour du Petit in 1712), we obtain a dilatation of all the arteries of the superficial parts of the head, and of all parts of the face, together with contraction of the pupil. As signs of hyperæmia, we observe increased size and pulsation of arteries, redness, increased heat, increased secretory action. If we excise the superior cervical ganglion of the sympathetic, we produce hyperæmia of the brain itself. In medicine we cannot resort to this radical means of paralyzing the vaso-motor nerves.

Of means to be used for this purpose, the most effectual is inhalation of the nitrite of amyl. The proper dose is from two to five drops.

If the remedy is to be administered by the medical attendant, it needs no preparation; five to ten drops may be poured upon a cloth or handkerchief, and a part or the whole of the quantity allowed to enter the patient's nostrils. In many cases the physician is away during the illness or seizure requiring the use of nitrite of amyl, and the nurse may give it safely, diluted in alcohol in such a way that a teaspoonful shall hold the proper dose.

The effects of amyl, apparent in a few seconds, consist in hyperæmia of the head and face, of increased heart action, and of redness and heat in the remainder of the body sometimes. From the condition of the face and external parts of the head, and from the sensation of fullness in the eyes and within the head, associated often with giddiness, we infer that there is hyperæmia within the cranium as well as without.

One indication for the use of this remedy we find in the condition present at the commencement of an epileptic convulsion. In the very earliest stage, during the occurrence of the aura, or the period preceding the aura, which is recognized in some patients, is the proper time to resort to this remedy. If used then, or at the moment the spasm of the vaso-motor nerves commences, indicated by the first signs of failure of consciousness, in many cases the attack can be averted; in others diminished in severity. Nitrite of amyl has also been used with success in angina pectoris, in asthma, and in various spasmodic affections involving muscular or vascular parts.

Chloroform has been used in the same manner, but its mode of operation is unknown, and it is much less effectual.

Another means is by over-stimulating the sympathetic nerves, but this means cannot be employed with any great degree of certainty. This is more theoretically than otherwise correct (*vide infra*, sub-class 2, *e*).

(*e*)–The means which produce functional hyperæmia are exceedingly numerous, embracing as it does all those agents which increase the activity of the nervous centres.

Hyperæmia of the nervous system occurs as the result of functional activity in the same manner as it is developed in the various organs of the body as the result of the same cause; such as the congestion present in the stomach and intestines and liver during digestion, etc. In this connection it is well to remember that it has been shown that increased temperature about the head is developed during exercise of the mental faculties, or by the operation of emotion.

The medicines referred to will be found in class B, sub-class 1.

(2) The second sub-class of A embraces all the means employed to diminish the amount of blood circulating in the nervous centres.

(*a*) Venesection.
(*b*) Decubitus.
(*c*) Compression of veins of the limbs.
(*d*) Cold.
(*e*) Those means which stimulate the vaso-motor nerves.
(*f*) Means which diminish the activity of the nervous centres.

(*a*)–There can be no doubt that blood-letting, if sufficiently large, will produce cerebral anæmia, for although physiologists still believe that the quantity of contents of the cranium is invariable, yet they to-day admit that the amount of intra-cranial blood may be reduced, serum taking its place about the vessels and in the sub-arachnoid space. I need hardly tell you that bleeding is out of fashion, and that we nowadays never use it. I would, however, say that with sufficient symptoms of sudden and violent cerebral hyperæmia occurring in a subject full-blooded enough not to be injured by the loss, I should bleed without hesitation by a large opening, and to impending syncope.

It is perhaps right for me to speak here of leeches and wet cups, in order to express my belief that these agents do not relieve hyperæmia of deeply placed organs so much by withdrawing blood as by irritating nerves, and thus acting upon the vaso-motor nerves of the affected parts. These means should therefore be placed in class D, with counter-irritants.

(*b*)–If the brain is the nervous centre to be affected, the decubitus should be with the head raised; or the patient should be placed in a chair with his legs and arms in a pendent position. This latter posture is useful in certain cases (rare, I believe) of insomnia dependent upon true primary cerebral hyperæmia.

In cases of spinal congestion, myelitis, or any affection of the spinal cord attended by congestion, place the patient upon his face. Relief may be secured by permitting the patient to occasionally lie upon the side, but lying upon the back should be almost entirely forbidden.

(*c*)–Compression of the veins of the limbs may be effected by means of ligatures. Formerly employed in convulsive affections, it has now fallen into almost complete disuse.

(*d*)–Cold, when used in a proper manner, undoubtedly has the power of diminishing the amount of blood in a part deeper than the skin. It must, however, be applied continuously if any benefit would be derived.

In the treatment of meningitis and all affections in which there is present hyperæmia, either primary or secondary, within the cranium, ice should be placed upon the head and at its base for considerable periods of time, carefully watching the effects in order to remove it at once if there is evidence that too much depression is being produced.

The same means may be used with benefit in intra-spinal inflammation and congestions.

Cold is also applied to the back of the neck and upon the spinal column with the object of acting, through the spinal cord, upon the vaso-motor nerves going to distant organs.

A word as to the means of using cold. You will hear Chapman's rubber bags highly recommended, but I suspect that their cleanliness and ease of application are what have caused their fame. It is best to apply the ice in block immediately to the skin, well guarded by cloths to catch the drippings; or pounded ice (with or without salt), inclosed in a cloth or bladder, may be used; or, lastly, the ether-spray douche may be employed.

(*e*)–The vaso-motor nerves may be directly stimulated by galvanism, causing contraction of the arteries supplied by these nerves and consequently anæmia of the parts to which these arteries go. This may be experimentally demonstrated for the face by galvanizing the sympathetic in the neck; for the fundus of the eye and the brain, by localizing the action of the current upon the superior cervical ganglion. The positive pole should be applied under the ear, in front of the mastoid process (small electrode), the other electrode be placed upon the fifth cervical vertebra; the current of from 4 to 8 Stöhrer's cells allowed to pass for one to two minutes.

Evidence of spasm of the cerebral arteries is obtained in the shape of vertigo developed at the moment of closing and opening the circuit. The vessels of the fundus of the eye have been seen to grow smaller under this application. Anæmia of a part of the brain is no doubt the inevitable immediate result of thus irritating the superior cervical ganglion, but a law of physiology stands in the way of our accepting this as a means to be used to meet the indication of diminishing the supply of blood. The law I refer to is that of never-failing relaxation after spasm, of exhaustion after stimulation. This law leads me to believe that *after* the primary cerebral anæmia produced as above described, there is a consequent equally inevitable relaxation of blood-vessels and hyperæmia produced. This is how I would justify the placing of this means, galvanization of vaso-motors, among those which increase the amount of blood in the brain (*vide supra*, sub-class 1, *d*).

Among medicinal agents, I know of few which will stimulate the vaso-motor nerves which are not medicines that should be embraced in another class. One exception, perhaps, is ergot.

Some recent observations seem to indicate that ergot diminishes the amount of blood circulating in the spinal cord and in many other tissues. It has been demonstrated that belladonna in small doses will accomplish the same thing. These two remedies, therefore, are quite generally used in the treatment of congestions and inflammations affecting the nervous centres. We are indebted to Dr. Brown-Séquard for their introduction into practice upon correct indications.

(*f*)–Diminution of the activity of the nervous centres may be brought about by mental and physical rest. By mental rest I understand not inactivity, but a cessation of the strain, intellectual or emotional, which has acted upon the patient. The merchant or professional man should diminish or cease altogether his work, whether in the way of actual labor or responsibility; he should resort to other occupations and to amusement. On the contrary, the emotional girl, or the insane subject, should be given work, physical if possible, in order to withdraw the attention from the contemplation of delusive sensual or pure emotional subjective creations. The operations of grief, of misery, of concentration in a diseased self, should be remedied by a variety

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of means (including amusements) which will suggest themselves to you. At all events, please remember that mental rest is not synonymous with inertia or idleness.

Physical rest is of great utility, and has been employed in locomotor ataxia with especial benefit.

Various remedies may be used for the purpose of diminishing the activities of the nervous centres, and these will be referred to in sub-class 2 of the next class, B.

Class B.

Means which Affect the Substance of the Nervous Centres.

(1) Agents increasing action of nervous centres—Excitants.

By nervous excitants I understand such means and medicines as produce (or can produce) an immediate stimulant effect upon the nervous centres, independently of any corresponding increase in vascularity. The certainty (specificity?) and rapidity of their action distinguish these medicines from tonics.

(A)—Cerebral excitants.	Alcohol, embracing wines, brandies, etc., Cannabis Indica. Belladonna, Opium, Ether, etc., Intellection, Emotional Influences.
(B)—Spinal excitants.	Strychnia, Brucia, Quinia, Cantharides, Exercise, (active and passive).

A.—CEREBRAL EXCITANTS.

It is very probable that alcohol acts upon the tissue of the brain and superior motor centres, as well as upon the circulation. I have already referred to the dosing of alcoholic drinks, and would only add that for the present purpose small quantities often suffice. In order to secure uniformity in the dosing of alcoholic stimulants, I should be in favor of using diluted alcohol instead of wines or brandy or whiskey. The only obstacles to the adoption of this practice are the prejudice existing against alcohol, and the fact that some stomachs bear wines and beer better than spirit.

Cannabis indica has been used in cases of melancholia and acute dementia, and in various neuralgic states.

Its specific effect is to excite the imagination. A sort of delirium is produced which, in most cases, is not accompanied by absolute unconsciousness, although the patient has the most extraordinary fancies, hallucinations, and delusions. It is this power of stimulating the imagination which has placed it among the remedies to be administered in apathetic cases. The success of the remedy, however, has not quite equalled the anticipations of theory.

In cases of cerebro-spinal mal-nutrition (*migraine, etc.*) its influence is beneficial.

Belladonna and opium are both stimulating to the brain when given in small doses, increasing intellection and producing hallucinations. When given in large doses they produce, besides, an after-effect of depression.

It is only upon some few individuals that this peculiar effect of belladonna is manifested; and many patients can take this remedy for a long time without complaining of any hallucinations or excitation of the imagination.

One of the dangers of administering opium is the temptation to continued indulgence in the use of the remedy for the sake of the intoxication which it produces. I would ask you always to bear this danger in mind, and to consider the responsibility attaching to the giving of opium as not ended until you are satisfied that your patient no longer uses the medicine. After treating a neuralgia, or other painful affection, with opium internally, or with hypodermic injections, you are to withdraw the narcotic very gradually—to wean your patient, as it were. I cannot conceive of a much greater cause of sorrow for a medical man than to have been the cause of the enslaving of a human being by the opium habit.

Ether produces intoxication, which is often very violent, when given by inhalation for purposes of anæsthesia. It is not ordinarily employed as a cerebral excitant.

Emotional excitement is not sufficiently employed as a means for affecting the nervous centres. It is seldom applicable where organic disease is present, but is more especially indicated where the mind is absorbed in contemplation of delusions, and in hypochondriacal and hysterical cases.

Cases of hysterical paralysis have been cured by an alarm of fire, or some other immediate danger. The pleasurable emotion of hope is very powerful, and its use makes possible the success of various quackish methods. In legitimate practice we should, I think, make greater use (within the bounds of truthfulness) of the potent emotions of hope and faith.

Many cases of melancholia have been benefited in a remarkable manner by getting them interested in some matter, either of love, or affection, or emulation. There are patients who have a certain amount of dementia, produced by the presence of some convulsive disease, such as epilepsy, chorea, etc. In these cases intellectual exercise is to be recommended and insisted upon; some intellectual excitement should be daily enforced.

B.—SPINAL EXCITANTS.

Nux-vomica, strychnia, and brucia are medicines which affect in an almost specific manner the motor parts of the spinal axis, stimulating it primarily. The result of over-doses of these remedies is spasm of muscles supplied by spinal nerves. In small doses these drugs act upon the same parts slowly, and improve their nutrition. The indication for the use of strychnia, etc., is the existence of simple exhaustion of the nervous centres, of spasm due to weakness and anæmia, of paralysis (inhibitory or reflex) not due to inflammation of the spinal centre.

We also make use of these remedies in some cases of functional nervous disorders. They are beneficial in cases of chorea and epilepsy, the morbid states grouped together under the name of "spinal irritation." In many of these cases the remedy (strychnia) should be given in the manner long ago pointed out by Brown-Séquard, that is, in doses necessary to obtain its physiological effects.

Bearing in mind that females are much more susceptible to the influence of these drugs than males, you should commence the giving of strychnia by doses of .0015 or .002, increasing rapidly to .004 or .005 three times a day for adults. There is not the same ratio of susceptibility to the influence of this remedy, with regard to age, as is seen in connection with opium and some other remedies, children bearing large doses well. Nux-vomica may be administered in .015 or .02 doses thrice a day, and the doses may be progressively increased.

It is better to use strychnia in solution. If given in pill, you will run the risk of the drug not having been carefully divided in the preparation of the pills; and the additional danger, which is much more likely to happen, that the pills will not be dissolved in the alimentary canal until an accumulation of them has taken place, which will develop results most undesirable. The necessity of using an acid to properly effect a solution of strychnia permits us to use diluted phosphoric acid, which is itself a serviceable remedy in the treatment of nervous affections; or we may use the various acid phosphates which are now offered to us in elegant preparations by druggists.

In cases of paralysis, strychnia may be used hypodermically, with benefit, .0015 to .006 being injected once a day, or once every other day. For this purpose I would recommend Barwell's solution at half strength, one drop of which contains about .001 of strychnia.

Quinia sometimes acts in a manner which entitles it to a place in this class of remedies.

There has been considerable doubt as to whether quinia affects the nervous system at all. There are, however, good reasons for believing that the phenomena of intermittent fever are of spinal origin. Quinia cures all forms of this disease almost with certainty. Another reason for believing that quinia affects the nervous centres is the fact that its administration aggravates spinal disease. According to Brown-Séquard, it acts as a poison to epileptics, and this effect will allow of only one explanation, namely, that the motor centres concerned in the production of the paroxysm are excited and rendered more susceptible by this remedy. Lastly, we have the general tonic effects of quinia, which cannot be denied.

Quinia will also stimulate the cerebrum, as is shown by the fact that intellectual exertion is more easy and free after its use. Dr. W. H. Draper, of this city, has given small doses of the medicine to two well-known clergymen with the result of restoring their power of extemporaneous speaking.

Cantharides excites the spinal cord, and seems to act more particularly upon its lower portion, manifesting its action by the development of symptoms especially connected with the genito-urinary apparatus. It is useful in cases of bladder paresis, of impotency, or simple genital inertia.

Exercise, both active and passive, is indicated in conditions of paralysis or paresis, or in simple spinal debility. Much of the passive exercise we make use of is obtained by means of electricity, which will be separately considered. Exercise should never be pushed to exhaustion; and I would have you bear in mind that exertion more easily produces exhaustion in the subjects of nervous diseases, and in the insane especially.

CLASS B.

SUB-CLASS II.

Depressants and Anæsthetics.

(a)—Cerebral depressants.	Cold, bromides of potassium and calcium, opium, hydrate of chloral, food, etc., chloroform, ether, etc.
(b)—Spinal depressants.	Conium, bromide of potassium, cold and food.

(a) Cold is to be employed in the same manner as when we wish to produce an anæmic condition of the brain. The inclination to sleep experienced by persons exposed to severe cold, such as encountered in the Arctic regions, is an illustration of the effect which cold can produce upon the nervous system.

The India-rubber bag sometimes employed in making applications of ice diminishes the intensity of cold applied, and is inferior to the bladder ordinarily employed (*vide supra*).

Bromide of potassium has usually been spoken of as acting through the blood-vessels, and causing their contraction and consequently anæmia of the parts they supply. Dr. W. A. Hammond and many others speak of the remedy as acting in this manner.

There has been some doubt expressed as to whether bromide of potassium is true hypnotic.

At present, perhaps, this is to be regarded as an unsettled question. For my own part, judging from my own experience and the testimony of others regarding bromide of potassium, I have been led to conclude that, when given in sufficient doses, it acts very powerfully as a cerebral depressant. In health, I admit, that no narcotic effects are obtained from a few doses of the medicine, but in cases of cerebral irritation, and insomnia from a variety of causes, its action is sure and immediate. In delirium tremens, for example, in such cases as are not complicated by degenerative liver and kidney disease, in the so-called sthenic cases, bromide of potassium given in doses of 4. or more every hour, until 12. or 16. have been taken, will, conjointly with semi-darkness and quiet, cut the attack short by producing sleep. In

1866–67, while House Physician to the New York Hospital, I had the opportunity of thus treating a series of cases of this disease with very satisfactory result. I say this while perfectly aware that several medicines seem to shorten the duration of delirium tremens, and that it is, under certain conditions, a self-limited disease.

In certain forms of insomnia seen in connection with fevers, the bromide of potassium may fail to produce sleep, because (?) of the continued influence of a blood-poison in keeping up the morbid state.

In the insomnia and delirium of pneumonia I have also seen speedy relief procured by 2.4 or 4. doses of bromide of potassium. In the simpler (?) conditions of sleeplessness caused by anxiety, over-exercise of the brain functions, emotional disturbance, this remedy usually acts well. Perhaps the most convincing proof of the action of bromide of potassium upon the cerebrum is to be had from observing the effects of the long-continued use of large doses of the remedy. A condition called "bromism" is set up, characterized by stupor, deficient memory, aphasiform speech, tottering gait, loss of facial expression, salivation, mucous irritation, papular skin disease, etc. Although we see this result more often in the course of the treatment of epilepsy, yet it may be developed in persons not having this disease. The mental state of brominized persons is not unlike that of patients with dementia.

Hydrate of chloral is, in my opinion, the best medicine that can be employed for producing sleep. It does not intoxicate, or disturb the digestive organs, as opium does. When chloral succeeds—and it almost invariably does succeed—it produces a calm sleep, not followed by any special disturbance of the system.

I would have you bear in mind that this remedy is a much more direct hypnotic than opium, and yet that it does not possess the power of preventing the perception of pain. Hence a rule to follow is to give chloral in cases of insomnia not connected with pain. The physicians to insane asylums in Europe and in this country have found in chloral an invaluable agent for giving rest to patients suffering under mania in any of its forms. They have also testified to the very great safety attending the use of the medicine, many patients getting large doses of it nightly for months and years. Chloral may also be used in cases where (*vide supra*) bromide of potassium is indicated, and the two may be given together. If you are to choose between these two drugs, use bromide of potassium if in addition, to the cerebral irritation there exists some physical excitement (spinal irritation); chloral in cases of pure cerebral disturbance. The theoretical view that bromide of potassium acts by causing anæmia of the brain leads to the withholding it in cases of cerebral anæmia and malnutrition. This I believe to be an error, since in my experience the occasional giving of bromide of potassium (and chloral hydrate) in such cases has in no way hindered the good effects of restorative medicines.

The dose of hydrate of chloral may be, for adults, 1. or 4. In females especially it is well to try a small dose on your first trial. A 1.2 gm. dose I believe to be a perfectly safe one for a man. The doses of 2.4 and 4. should be used in cases of acute mania or severe delirium.

The conjoint use of bromide of potassium and chloral is very satisfactory. Four gms. of bromide of potassium may be administered in the afternoon, followed at bedtime by 1.2 or 2.4 of chloral.

Concerning the use of opium and its constituents in diseases of the nervous system I believe that I need say very little. I would have you always bear in mind the exceedingly unpleasant way in which this remedy affects very many females, producing insomnia and, later, vomiting and constipation. For these susceptible individuals (a few males must be included) some of the liquid compound preparations of opium, McMunn's elixir, the liquor opii comp., may be substituted for morphia or simple opium. In a very large number of cases of nervous disease we seek to relieve pain by the use of morphia. In such cases it is best administered hypodermically, in the form of strong solution of the sulphate or acetate or muriate. I must admit that my own patients seldom receive any but the sulphate of morphia injections, in the shape of Magendie's solution (1. to 30.), .60 of which are equivalent to .02 morph. sulph. I am, however, in the habit of adding a little atropia, .0006 or .0008 to each hypodermic injection, for the purpose of palliating the nauseating and constipating effects of the morphia. My solution of atropia is composed of atropia .06, distilled water 20. : .06, containing .0002 atropia. I have employed this combination since 1867 with great satisfaction.

Food is a depressant, because it remedies the morbid irritability produced in the brain in cases of exhaustion, whether from hemorrhage or over-work.

Sometimes the effect of food is very marked indeed. Many of you have doubtless experienced a sensation of exhaustion, accompanied by headache, perhaps severe, which has been entirely relieved by taking a good meal. The headache perhaps has been made to disappear, even before the meal was completed, and you have felt immediately refreshed, and that without wine or other alcoholic stimulant.

In many cases of delirium and mania the effects of the persistent giving of nutritious food are evidenced by rapid improvement and cure.

b. Spinal Depressants.—Conium is the typical medicine of this class, and seems to be the direct antagonist of strychnia. It acts by paralyzing the spinal motor centres, from the nucleus of the third nerve down. This remedy can be used with benefit in spasmodic affections.

In small doses it produces a paretic condition of the spinal axis, indicated by partial ptosis, strabismus, or double vision, weakness of the knees; and the arms may become slightly paretic. These symptoms appear within an hour after the medicine has been taken. By the administration of larger doses, almost complete akinesis is obtained, which may last for half an hour or an hour, but is not dangerous.

The remedy should be given only once a day, and in many cases to the extent of partially paralyzing the patient.

A reliable preparation is the English *succus conii*, which may be administered in doses from 8. to 24. cc. once a day. An equally good form of conium is Squibb's fluid extract. I usually prescribe this and give it in doses of 2.5 – 4. cc. Dr. John Harley, of London, has done much to give us a clear notion of the action of this remedy, and he has found it useful in chorea, epilepsy and other spasmodic diseases; he points out the necessity of obtaining the physiological effects of this and other drugs when we wish really to do good in affections of the nervous system. In epilepsy I have employed the fluid extract of conium in combination with bromide of potassium with good results.

Bromide of potassium also acts directly upon the spinal cord as a depressant. It lowers the activity of the motor tract (though in a lesser degree than conium) and diminishes reflex excitability. Its use is consequently indicated in all affections in which reflex action is abnormally great, and in many such we obtain immediate and good results from its administration. In various forms of convulsions, the eclamptic attacks of children, of pregnant and parturient women, bromide of potassium does good. Morbid excitement of the lumbar part of the spinal cord, as evidenced by nymphomania and satyriasis, is often relieved by this drug. Some forms of vomiting (in pregnancy, after inhalation of ether, etc.), spasmodic states of various sphincters, are also cured by it. It is in the treatment of the great neurosis epilepsy that this medicine is the most employed, and it is concerning its usefulness in this affection that great discussions have occurred. The use of it in epilepsy was begun by several physicians about the same time, but Drs. Brown-Séquard and Laycock were the first to call attention to it. The generally received opinion is that it is the medicine which possesses more power than any other over epilepsy; that in the majority of cases the frequency and severity of the seizures are very much diminished while the medicine is being taken (the symptoms soon reappearing if it be discontinued); and that a case here and there may be cured by its use. In hospitals with large numbers of epileptic patients the effects of giving and withholding the bromide are very strikingly in favor of the utility of the drug. For my part I wholly accept this as a correct estimate.

There are a few general rules to be observed in the treatment of epilepsy by bromide of potassium.

In the first place enough bromide of potassium (and other bromides if you please) should be given to reduce the reflex function and keep it below the normal standard. A test of the sufficient action of the medicine lies in the reaction of the palate and fauces to irritation; a diminution or abolition of the well-known reflex movements of these parts indicating diminution of the reflex excitability. Another general rule is to give more of the medicine at night than in the day-time; a direction of great value, for which we are indebted to Dr. Brown-Séquard. We usually give three day doses, and a dose in the evening twice or thrice the size of the day dose. A third and most important rule is to administer the salt in a perfectly continuous way for months and years. Dr. Brown-Séquard has known patients remain without seizures for two years while taking his prescription for mixed bromides, having a return of convulsive seizures in a short time after ceasing the medication. I have seen similar though less striking results.

There are a few epileptics who cannot tolerate the bromides; who become easily "brominized," and whose attacks are made worse by these drugs. These paradoxical patients are very rare, I believe. While recognizing the great value of Brown-Séquard's compound bromide solution I more commonly employ a simple solution of bromide of potassium, giving .30 three times a day, and 1. or 1.20 at bedtime, at first in adults. Children require relatively very large doses of the bromides, and, contrary to what is sometimes taught, I see in anæmia no counter-indication to the use of the remedy, nor do I believe that any law can be laid down for the giving or not giving of it, from observations upon the retinal circulation. Such a view is based only upon belief in the more than doubtful physiological theory that cerebral hyperæmia and anæmia are usually prime factors in the pathological state called epileptic.

CLASS C.

Tonics and Restoratives.

By restoratives I understand, with Headland, those remedies which restore to the system an element, diminished by a disease, or whose diminution causes a disease. A few of this class are especially useful in diseases of the nervous system, two chiefly, —phosphorus and fats.

I would not be willing to admit that there is any defined morbid state of the nervous system which can be shown to depend upon a diminution of the phosphorus, which is so important an ingredient of nerve tissue, yet I am the first to recognize that in some nervous diseases much phosphorus is excreted, and that in very many of them much benefit, even to a cure, is obtained by giving phosphorus. In practice the various phosphates, the acid phosphates, the hypophosphites, etc., if they do good, do so very slowly, and are hardly to be used in the treatment of serious cases, except as adjuvants. Phosphorus itself may be administered in the form of the officinal oil, or in the non-officinal solutions (Thompson's), and as phosphide of zinc. The dose of phosphorus ranges from .0016 to .005; that of zinc phosphide from .01 to .03. In administering this powerful remedy, please bear in mind that some organizations are very susceptible to its toxic influence. In cerebral mal-nutrition, in neuralgia, in "spinal irritation," in hysteria, and in varieties of paralysis, this drug is of the highest value.

Fatty food and cod-liver oil are indicated in the conditions which demand phosphorus.

Among the tonics the chief are strychnia, arsenic, zinc, iron, quinia and cold. Cold should, for its tonic effect, be applied only for a short period of time. This may be done by sponging, the shower-bath, cold compresses, the cold sheet and sea-bathing. A corresponding reaction follows, which consists in hyperæmia and improved nutrition. (*Vide infra*, counter-irritants.)

We are unable exactly to explain why strychnia, or arsenic, or zinc, should benefit the general nutrition, but daily experience teaches that these remedies are invaluable in many morbid states of the nervous system.

Strychnia, in cases of irritability, of hysteria, spinal irritation, and in some palsies, may be given in small doses for long periods of time; doses of .0016 or .002. The action of arsenic is often marvellous in chorea, and very satisfactory in other nervous diseases. Fowler's solution is the arsenical preparation most commonly employed, and in chorea it should be dealt out with no sparing hand; doses of from .30 to as high as 1.2 cc. being well borne. The oxide and lactate of zinc have been much used in states of exhaustion of the nervous system, after sexual excess, or in chronic alcoholism, and in epilepsy. In the nervous states caused by alcohol the zinc oxide proves almost as satisfactory as arsenic in chorea. I have usually employed it in combination with extract of nux-vomica. Since the introduction of potassium bromide into general use, the zinc treatment of epilepsy has fallen into (perhaps unmerited) disuse.

Quinia, in moderate doses, would seem to act as a tonic. It is possible that it does so by causing more food to be taken and digested, yet from the immediate improvement in well-being and in cerebral activity which many experience while taking it, I am inclined to the opinion that this remedy does exert a direct effect (restorative ?) upon the nervous centres.

Iron I have not spoken of, because it does not especially affect the nervous system. It improves the condition of the blood, and, by so doing, cures morbid states of the nervous system (neuralgia), which depend upon anæmia or chlorosis.

Perhaps it were well that I should here speak of two so-called specific modes of medication, very useful in nervous diseases, and which would not otherwise enter the classification offered you. I refer to the treatment of constitutional syphilis, which often gives rise to morbid states of the nervous system. Nearly all of these morbid states are a part and parcel of the third (tertiary) stage of syphilis, depending upon affections of fibrous tissues of bones, of blood-vessels, and upon the presence of gummata in the nervous centres. I would only speak of one point in connection with this subject, and that is the necessity of using the iodide of potassium in really effectual doses, giving from 4. to 24. a day until improvement takes place. You will be surprised to see how a patient with tertiary syphilis will gain while taking 16. of iodide in the twenty-four hours, after having resisted smaller doses. No unpleasant symptoms attend the taking of large doses, provided that restoratives and good food be also given.

The other specific medication is the treatment of malaria in its original form by quinia. Malarial neuralgia is a well-known and an obstinate affection. It may be cured by quinia in the usual way; but a much more rapid removal of the disease is obtained by using the quinia in the shape of hypodermic injections over the affected nerve. We then obtain the specific effect of quinia on the nervous centre and counter-irritation on the nerve. Nearly seven years ago* I published the results of my experience with this medication in the New York Hospital, giving full details of the practice. As a general rule, in nervous disorders dependent upon malaria, do not err by giving *too little* quinia.

CLASS D.

Counter-Irritants.

By counter-irritation is understood a localized irritation, which modifies the nutrition or mode of activity of another part than that irritated, either just beneath it, or at a distance from it. Counter-irritants almost always act through the spinal cord, and their mode of action is exemplified by a morbid process that takes place as a consequence of severe burns. If a limb or part of the body be scalded or charred, the patient is not in danger merely because of the shock of the injury, or of the exhausting discharge accompanying the healing of the burn, or of the pyæmia which may take place during the continuance of this suppuration, but he is also likely to suffer from certain visceral complications which occur in parts bearing a definite relation to the burn. Brown-Séquard demonstrated many years ago that by cutting across the spinal cord, above the origin of nerves going to the burnt part, no visceral lesions occurred; thus proving that these lesions were set up by a morbid state of the spinal cord, produced by the burn. The burn corresponds to our counter-irritation, and the altered nutrition of the viscera to the distant effect produced. In the one case a morbid process is induced; in the other a beneficial change, not yet understood by us, is determined.

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* On the Subcutaneous Use of Sulphate of Quinia in Cases of Malarial Neuralgia. New York *Medical Journal*, 1867, p. 402.

The alternate application of cold and heat does much to improve the nutrition of paralyzed parts, and is used for many special purposes.

Brown-Séquard has recommended the use of these means to prevent the formation of bed-sores, and to heal those which may be already formed.

The method consists in applying an ice or snow-poultice over the part for five or ten minutes, following this by the immediate application of a hot poultice. This should be done once a day over the parts threatened with the formation of bed-sores.

When the same thing is done twice a day to sores already formed, as a rule, sloughs come away, circulation is improved, and granulations will soon spring up. I have seen huge bed-sores healing under this treatment while the patient was failing.

The actual cautery is a means of the utmost value in the treatment of nervous affections. It is one, however, which has fallen into very great disrepute in consequence of the manner in which it was formerly employed. A burn sufficient to produce suppuration is not necessary, surface-irritation being what we seek to produce.

The theoretical view that surface-irritation is most useful, has, like several others brought forward in this lecture, a true physiological basis. It rests upon the well-known law that the terminal nervous twigs, and their special terminal bodies, are more irritable than nerve trunks.

Brown-Séquard has revived the use of the actual cautery; and by means of his form of cautery we are enabled to produce much skin irritation, with little pain to the patient, and no subsequent annoyance from sore surfaces.

The cauterizing iron which he employs is tipped with platinum. The platinum never gets rough by ordinary heat, as does iron, and there is, consequently, a smooth surface to come in contact with the skin every time the cautery is used.

The white-heat is necessary in using the actual cautery, because it produces the maximum degree of irritation to the nerve, with a minimum of pain.

The cautery thus heated (in a coal fire) should be rapidly drawn over the part selected, four or more strokes, of from one to six inches in length, being made. This can be done in a very few seconds, and in many cases no real pain is experienced by the patient. The strokes remain as reddish-brown welts, which are quickly surrounded by a zone of hyperæmia, which is sometimes immense. A moderate degree of burning pain is felt for twenty minutes or two hours after the application. Sores never result from the burning, and not once in twenty strokes have I ever seen blisters arise. The epidermis is cast off dry in a few days, leaving a brownish stain which passes off wholly in a short time. The cautery can be used upon the face with perfect safety. I have made use of this most valuable means in women, and children thirteen years old, without anæsthetics. The disagreeable ideas connected with the words "burning" and "cautery," in the minds of physicians and of patients, are the barrier to the more general use of the platinum cautery.

Setons have so much gone out of fashion that I need hardly stop to enter a protest against their use. It is very doubtful if a suppurating sore produces more irritation than the cautery or blisters, and there are grave objections to using setons.

Blisters are invaluable means of producing counter-irritation. Let me ask you to use them in such a way as not to produce suppuration; repeat the blisters and heal the blebs as soon as possible. Great good is often obtained from a series of small blisters.

Dry cupping is beneficial in many spinal affections. The number of cups applied should be large—from ten to twenty. It is not necessary to use the cups immediately over the seat of the disease. Ten cups may be applied upon the back, and ten in front, with the same benefit as if all were applied behind.

It is a matter of doubt whether wet cups act better than, or in a different manner from, the dry. The degree of skin irritation produced by the process of wet cupping is certainly very much greater than in that of dry cupping; and it cannot be positively denied that the loss of blood is useful. In various inflammatory affections of the cerebral and spinal meninges, and in some functional disorders, wet cups are still used.

Sulphur baths do good by the irritation they produce upon the surface, rather than from any effect of the sulphur upon the system generally. These baths may be employed liquid or in the shape of vapor. For liquid sulphur baths, the sulphuret of potassium is dissolved in water, and the patient soaks himself awhile in the solution. A better application is obtained by vaporizing sulphur in a chamber into which steam is admitted, the patient's head being kept out of the bath, of course. A stay of ten or twenty minutes in this atmosphere produces a great deal of cutaneous irritation, which, in time, leads to a decided desquamation. Some of the natural sulphur spring waters are also used for bathing purposes, but are much less effectual. In spinal diseases, particularly posterior spinal sclerosis (locomotor ataxia), series of such baths have done great good. They are also useful in lead poisoning. Many medicated baths likewise act by producing extensive irritation of the skin.

Class E.

Electricity.

We employ all three forms of electricity in medicine, Franklinism or statical electricity, Galvanism (or Voltaic electricity), and Faradism or induction electricity.

Any one of these forms may be used in the treatment of diseases of the nervous system in a rational or an empirical way.

The rational applications of electricity are to produce muscular contraction (passive exercises) and to affect the nerves of sensation (irritation, sedation.)

What I may call the empirical use of electricity is when we attempt, by means of it, to modify the mode of activity of deeply placed organs, such as the brain, spinal cord, and the ganglia of the sympathetic.

It would be out of place for me to attempt, in a few words, to speak of the special application of electricity. A great deal has been written about this matter in the last few years, and I am glad to say that there are a few small books on the subject which I would urge you to study. I need only say that within certain limits, corresponding with our physiological knowledge, I have the greatest faith in the power of Faradism and Galvanism as remedial agents, and that I use them a good deal.

CLASS F.

Hygienic Means, etc.

The cases of nervous disease which are strictly speaking active, due to hyperæmia or overaction of the nervous centres, I regard as being exceedingly rare. Consequently, I regard the immense majority of cases as requiring a supporting diet and such hygiene as shall tend to improve nutrition.

There are very many points to be considered in this connection.

In the first place, many paralytics suffer from slow digestion and constipation. The diet of such patients should consist of such articles of food as leave little detritus, as meats, fish, eggs, milk and fats. Vegetables and starchy or sweet articles should be allowed sparingly.

In the second place, the bladder in many cases (spinal palsies) requires to be emptied by means of the catheter. In such cases you cannot exercise too much gentleness and care in introducing the instrument, for fear of setting up cystitis, or of aggravating one already present.

Thirdly: In many spinal cases there is immensely exaggerated reflex activity of the spinal centres; spinal epilepsy is set up by the contact of the bed-clothes, your hand, etc., with the palsied limbs, and an overloaded bowel brings about attacks which seem spontaneous. By strict orders to the nurse and by the help of mechanical contrivance you can reduce these spasms to a minimum. If one occur, recollect that you can stop it by forcibly flexing one or both great toes, as indicated by Brown-Séquard.

Fourthly: I would ask you always to bear in mind that bed-ridden paralytics are peculiarly liable to fatal attacks of bronchitis, broncho-pneumonia, and pneumonic phthisis. Remembering this, you will give directions to obviate all that which might expose your patients to such chest complications.

Fifthly: Inasmuch as bed-sores are likely to occur among the complications of nervous diseases, a few words may be added to what has already been said concerning their prophylaxis.

It is important to make every piece of clothing beneath the patient smooth. Consequently, a pretty hard mattress or water-bed is the best for him to lie upon. Special care is to be taken that the under sheet does not get wrinkled and drawn into folds; its ends may be fastened down by tapes. The shirt worn by the patient should be kept smooth under him, and perfect cleanliness must be enforced. Special attention to these apparently small matters will in most cases be sufficient to prevent the formation of eschars.

In case a bed-sore has formed, the best course to pursue is to clean away all necrosed skin and connective tissue (tow-like shreds) by means of forceps and scissors, and then to use the ice and poultice treatment as above detailed. When healthy granulations spring up, ice poultices are still useful once or twice a day, to be succeeded by ointments, or adhesive plaster strapping.

Sixthly: In the matter of coffee, alcoholic drinks and tobacco. You will hear physicians asking their patients to give up the use of these articles simply because they have a disease of the nervous system. I am afraid, gentlemen, that this is very illogical. For my own part I do not proscribe these luxuries unless there is evidence that their use has had something to do with the development of the morbid state. Usually I do not at all interfere with the use of coffee, and ask that a less quantity of alcoholic drinks and tobacco should be used. The worry and nervousness consequent upon the giving up of an established habit is worse for the patient, in my opinion, than the moderate use of the above-named articles.

Let me close this rather fragmentary lecture, by calling your attention to something which is not wholly extra-medical. I allude to the care of your patients' spirits—their emotions and fancies if you will. Never let *alarm* be one of your medicaments. If necessary, in order to bring about the reform of bad habits, lay the picture of consequences before your patient truthfully, but not in the language of exaggeration. Even in hysterical cases you need not be so frank as to hurt your patient's feeling: it will often do if her relatives know precisely what you think of the value of the symptoms. In cases of mental disorder, pray do not forget that even very maniacal or melancholic patients attend to and remember all your words and actions, and will treasure any kind and careful behavior of yours as well as bitterly recall any unkind or hasty phrases and acts. As a general therapeutic rule, I would have you be as anxious to avoid wounding the sensibilities of your poor or wealthy patients as of injuring their tissues.

AN OUTLINE OF THE PHYSIOLOGY OF THE NERVOUS SYSTEM.*

GENTLEMEN:—In thinking of a subject to present to you in an introductory lecture, it occurred to me that a good one would be a sketch, very much condensed, of the anatomy and physiology of the central nervous system, to serve as a basis upon which we can, during the winter, build up a knowledge of diseases of the brain. I shall be very brief upon the individual heads of the lecture, wasting no time upon matters which are not capable of bearing a logical relation to pathology.

Before entering upon the subject proper, I wish to say a few words, intended to give you a clearer idea of the relation of medical to general knowledge. I mean to warn you against the error which it must be said some of the books in your hands tend to strengthen and perpetuate, viz., the separation of physiology from pathology, and the confounding of the latter with pathological anatomy. If we look at the matter from an extra-medical or philosophical point of view, we are led to classify our knowledge of the animal body into two categories: statical and dynamical knowledge. In other words, we study the animal organism and its parts in a state of rest, and in a state of activity; we observe the form and constitution of parts, and determine their properties and functions.

The study of the human body and its parts in a state of rest is called anatomy, in the broad sense of the word, making it to include chemical and microscopical analysis. Anatomy is spoken of as normal (with many subdivisions) when it describes the healthy parts and tissues; as pathological or morbid when it treats of the changes produced in tissues and parts by disease.

Our dynamical knowledge, embracing the study of the properties and functions of the parts we have anatomically analyzed, is called physiology; and this must, like anatomy, be separated into two great subdivisions—physiology, strictly speaking, and pathological (or morbid) physiology or pathology. The activities we observe in abnormal (pathological) states are not different in kind from those which pervade the organism in health; nor are the laws of derivation and utilization of these activities different from the laws which operate in the normal body. By close analysis we find that in diseased states, as in health, physical and chemical laws govern the frame, and that essentially the results of their operation are the same. It can, I believe, be demonstrated with all but mathematical exactness, that the great law of conservation and correlation of force is supreme in the disordered and in the perfect animal organism. There is and can be no entity of disease, no demon. Even therapeutics can be brought into accord with this conception of medical knowledge; for our medicines and remedial agents are only means by which we aim to act upon the human body in such a way as to modify the shape or constitution of its parts, and to alter the mode of activity of its tissues and organs. And although the *modus operandi* of most medicines is obscure as yet, I think it not rash to say that medicinal agents do not act in any supernatural or occult way, but by the laws of physics and chemistry.

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* A lecture introductory to clinics upon Diseases of the Nervous System, delivered at the College of Physicians and Surgeons, New York, on Saturday, October 3d, 1874. Reprinted from the *N. Y. Medical Record* of Dec., 1874, vol. ix.

The study of the physiologist, and of the pathologist as well, begins with the nutrition of simple tissues, single cells, or even masses of protoplasm; and upon such a study, as a basis, there is built up the more complicated and elaborated knowledge of the activities of the human body which the practical physician needs so much at the bedside.

A consideration of what I have said will, I trust, lead you to admit two propositions: first, that we physicians are in reality naturalists, studying what we call disease by natural and scientific methods; second, that the most excellent physician must be the man who (contrary to a vulgar prejudice), together with a practical turn of mind and a sympathetic nature, has the greatest amount of scientific knowledge at his command.

I have thus apparently gone out of my way in order to help you in viewing this lecture as a logical whole, an imperfect attempt to give you a guiding plan in your studies of diseases of the nervous system.

The subject of the lecture is naturally divisible into three parts.

1st. A study of the elementary parts of the central nervous system—their anatomical attributes and physiological properties.

2d. A study of the organs and apparatuses in the central nervous system, and their functions.

3d. A summary of the chief modifications of the properties and functions which constitute the symptomatology of disease.

1. The part comprising the nervous centres may be classified as follows:

A. Bindweb (neuroglia).
B. Blood-vessels and lymphatic spaces.
C. Nerve fibres { myelinic.
  { amyelinic.
D. Nerve cells (ganglion cells).

A. The bindweb, gentlemen, is the framework in which lie the strictly nervous elements of the nervous system; it incloses and supports nerve fibres, nerve cells, and blood-vessels. Its histological composition is that of a fibro-connective tissue, according to the latest observations. In it we meet with bundles of connective-tissue fibres, with exceedingly delicate fibrillæ of fibrous tissue, and with more or less altered cells lying often at the point of meeting of the fibrillæ. The fibrous fibrillæ are more immediately in relation with the nerve fibres and cells, while the connective tissue, constituting the septa and the cortical layer of the spinal cord, are united at the periphery with the pia mater, or innermost membrane of the three surrounding the brain and cord. The bindwebs of the brain and spinal cord are similar in kind, but the cerebral fibro-connective tissue is much the more delicate. The statements that there is a "granular matter" in the neuroglia, or that it is a sponge-like tissue, I believe from personal observations to be based upon inexact examinations. At any rate, it suffices for our purpose to know the bindweb as a form of connective tissue, containing nuclei which may be the starting-point of proliferation changes tending to truly pathological, or to reparative changes.

B. The blood-vessels of the nervous centres are very abundant, and are in many respects peculiar. The capillaries are by far more numerous in the gray matter of both encephalon and spinal cord than in the white substance. The larger vessels all reach the nervous organs through the pia mater. The rarity of anastomoses between the arteries in the cord and brain is a most striking feature, and is closely related with pathological processes. The non-capillary vessels are remarkable for the relatively extreme development of their muscular coat. The greatest peculiarity presented by these vessels, however, consists in their having an outer sheath, constituting a canal separated from the vessel by a small amount of fluid. All the vessels of the cerebrospinal mass are thus inclosed. These so-called perivascular canals are made up of very delicate connective (structureless?) tissue, and contain a fluid very analogous to lymph. The perivascular canals are an extension into the parenchyma of a vast system of lymphatic spaces which surround the nervous centres, the so-called sub-arachnoid space in the meshes of the pia mater around the brain and spinal cord. This arrangement has been known for several years; and we now know, by the recent investigations of Ranvier and Axel Key, that the same sub-arachnoid or lymphatic space extends outward with every cerebral and spinal nerve to its termination. You should imagine the nervous system—brain, spinal cord, and their nerves—as floating in lymphoid fluid, contained in a delicate connective-tissue envelope. Even in the eye-ball and internal ear these lymph spaces have been demonstrated. We are only beginning to appreciate the bearing of these important anatomical discoveries to pathological processes. It has been stated that nerve cells, in the cerebral convolutions especially, are surrounded by a similar lymphatic space—an extension of the one described—but this is, I am convinced, an error of interpretation, the space seen having been produced by the shrinkage of the cells under certain modes of preparation.

c. Nerve fibres. Several classifications of nerve fibres are in use, but the simple one of two classes—myelinic and amyelinic—will suffice for us physicians. But the term myelinic nerve fibre is meant a complete nerve fibre, one consisting of three parts—a central body or axis cylinder, a surrounding mass of fatty substance, the myeline, and a structureless enveloping sheath (the membrane of Schwann). In some parts of the nervous system the last element is absent. The amyelinic fibre is simply a naked axis cylinder. You see from these definitions, that the essential part of the three is the axis cylinder; the other two, which may be called insulating, are superadded. The amyelinic fibres are met with in the gray substance mostly, but are also abundant in the sympathetic nerves. We find myelinic nerve fibres, without the membrane of Schwann, and varying infinitely in size, in the white columns of the spinal cord, and in the white substance of the encephalic mass, constituting the bulk of these parts. There are also such fibres, though minute ones, in the gray substance of the cord and brain. The peripheral parts of the nervous system, all the cerebral and spinal nerves, and a part of the sympathetic, are made up of the second variety of myelinic fibres—those which have the three parts.

A peculiarity of nerve fibres is that they extend independently of each other, *i.e.*, not anastomosing, from their origin to their distribution, from nerve-cells in the central nervous mass to peripheral organs; and this isolation of nerves, together with their insulation, explains a physiological attribute to which I shall call your attention. I should add that forty years ago it was believed that nerve fibres extended from the brain to the outer parts of the body, and that the spinal cord was chiefly a bundle of nerves. Progress has strongly tended to take this supremacy away from the brain, to show us the importance of the spinal cord as a centre; and we now believe that nerve fibres once in the spinal cord run only a comparatively short distance before uniting with nerve cells.

D. Nerve cells are the noblest elements of the nervous system—those which possess the power of generating force in the modern acceptance of the phrase, *i.e.*, the property of evolving nerve force out of chemical activity. They are found in greatest number in the brain, spinal cord, and sympathetic nervous system—aggregations of them constituting gray matter or ganglia. Nerve cells, wherever found, consist of organized matter, call it protoplasm if you will, not inclosed in any cell membrane, sending out prolongations or processes of various shapes and lengths, containing a globular body called the nucleus, which itself incloses a smaller body termed the nucleolus. These nerve cells vary in size, in shape, and in the number of their processes. The largest ganglion cells are met with in the anterior gray horns of the spinal cord in its lumbar and cervical enlargements. In the ox, cells taken from these localities are almost visible to the naked eye. In the floor of the fourth ventricle (medulla oblongata) and in certain parts of the base of the brain large cells are also found. The smallest cells occur in the gray matter of the cerebral convolutions, and in parts of the medulla oblongata. As regards shape, four subdivisions may be recognized:

polyhedral, pyramidal, oval, and round. The first are met with in the same location as the largest cells; the pyramidal are nearly restricted to the cerebral convolutions; oval cells are found in the median and posterior parts of the gray matter of the cord, and in the medulla; round cells in the sympathetic and in the ganglia of the posterior roots of the spinal nerves. With respect to the disposition of their processes, cells are distinguished as multipolar, bipolar, and apolar. I do not believe in the existence of apolar nerve cells. Under the name of multipolar cells we include a majority of ganglion cells; those of the cerebral cortex exhibiting from three to six processes, the large cells of the medulla and spinal cord showing a great many—eight, twelve, or more. Bipolar cells are rarely seen in preparations from the human nervous centres. The separation of cell processes into two classes was an important progress. A multipolar cell sends out one stout, thick process, which extends a long distance, not subdividing, ultimately forming, with the addition of myeline, a nerve fibre. The same cell also throws off an indefinite number of processes of unequal size, which rapidly subdivide, growing smaller and smaller. The former is the "cylinder axis process;" the latter are the "protoplasmic processes," and their destination is unknown. It has been claimed that the number of processes and the size of cells afford an indication of their special functions, but that is not believed to-day. I should add that the communications between cells which you will find figured in books do not exist; and that the communication of nerve fibres with nerve cells has been demonstrated, though a very few times.

These elementary structures, while alive and forming a part of the animal body, possess certain physiological properties, some common to all, others the special attributes of individual parts. The most common of these physiological properties is that of being osmotic, *i.e.*, of allowing fluids to pass through them. This property is possessed in a very high degree by capillary vessels, by arterioles, venules, and the perivascular sheaths—the escape of material from them to the tissues, and *vice versâ*, being very rapid and free. The bindweb, also, has the osmotic property in a high degree, and thus serves as an aid to nutrition as well as a mechanical support. Nerve fibres and nerve cells are undoubtedly osmotic, but to a much less degree. Wherever it exists, this important process is under the same laws; it is only possible when liquids of different density are on either side of the membrane; its rapidity is increased by motion, pressure, heat, and chemical action, all of these being found in the living nervous centres.

Chemical changes of a very complicated sort are going on constantly in the cells and nerve fibres of the central nervous system, constituting the essence of nutrition. In a state of health the acquisition of new material by the tissues is so balanced with the separation of effete matter, that, in spite of great internal activity, the parts are maintained in a uniform (not mathematically equal) condition. We should never forget that this chemical action in myriads of parts cannot take place without producing other correlative effects, such as nerve force, heat, and electric currents.

Nerve fibres possess certain special physiological properties. In the first place, they conduct the impressions they receive, in both directions, from the central organs to the periphery and *vice versâ*. This conduction is not by any means instantaneous or even very rapid, as it takes place in isolated nerve fibres at the rate of less than 55 meters per second, whereas the speed of electricity is 428,000,000 meters; that of light, 277,000,000; that of sound, 306; that of a cannon-ball, 509. In the living body the rate of transmission is from 36 to 40 meters. This conduction is done, furthermore, in a perfectly isolated way by individual nerve fibres; there is no interference between fibres on their way to and from the central organs. Nerve fibres are excitable, that is, respond to stimuli—mechanical, chemical, and electrical—by motor manifestations or by sensations when the motor or sensory filaments are experimented upon. This excitability is quite independent of the nervous centres, and is inherent to the nerve, as is shown by the fact that a nerve continues to react to stimuli for three days after its separation from continuity with the nervous centre.

Nerve cells have properties whose existence we learn in part through reasoning by exclusion, after having ascertained the properties of nerve fibres, and in part by direct experimentation: In the first place, certain nerve cells have the power of furnishing force (motor impulse) to nerves and muscles; this is called motricity by some authors. Another property of nerve cells is sensitivity, that is, the property of transforming impressions received from without by and through the sensory nerves into a sensation. That nerve cells possess a power over the nutrition of parts non-nervous, we now incline to believe; but we hardly yet dare name and define this property. But nerve cells have, I believe, yet one physiological property, viz., that of retaining impressions made upon them; a property for which I now propose the term retentivity. I have for some time believed that nerve cells (and other cells to a degree) do in all parts what it is acknowledged they do in the cerebral convolutions—they possess memory, or the property of registering or retaining impressions. That this is probable is shown by the fatality of numerous actions occurring a second time and oftener. The occurrence of a sensation will give rise to a flow of ideas associated with the sensation, and this under normal conditions will be repeated whenever the sensation is renewed. An action of the class called reflex or sensori-motor is, after its first performance, fatally repeated whenever the same initial sensory irritation occurs. A bolus of mixed foods passing down the alimentary canal provokes in a necessary or fatal way the action of various muscular, vascular, and glandular organs. The well-known experiment of placing a drop of acid near a frog's anus, after decapitation, illustrates my view of the possession of memory by the nerve-cells of the spinal cord; for in this experiment the hinder legs of the animal are drawn up and moved in an apparently intelligent manner, in such a way as to remove the irritating acid. Three years ago, in spring lectures given here, I explained this phenomenon by saying, that the frog having during its life often performed this act for the same purpose, its occurrence after cerebral death takes place by necessity, because the same sensation is transmitted to the spinal cord. Additional proof of the correctness of this theory is to be obtained from a study of the mode of acquisition and retention of complex co-ordinate movements, such as walking, dancing, piano-playing, etc. Motricity, sensitivity, and retentivity are therefore the chief special physiological properties of nerve cells.

Let us now, gentlemen, resume our statical, or if you please, anatomical study. The various nervous elements which I have sketched for you are combined in the living body in such a way as to constitute organs and apparatuses. The term organ I would apply to such parts as are the seat of performance of relatively limited and less important functions, while by apparatus I understand a combination of organs serving for the evolution of important and comprehensive functions. The spinal cord, medulla oblongata, pons Varolii, cerebellum, and cerebrum may be named as the central nervous organs, made up as follows: The cerebrum consists of a superficial or cortical layer of gray matter, *i.e.*, of tissue made up of nerve cells, and both kinds of nerve fibres (myelinic and amyelinic), arranged in a somewhat complicated way. This gray matter rests upon the white substance composed of myelinic fibres, which extend downward and inward to certain basal gray bodies or ganglia, the optic thalamus, and the corpus striatum; or the white substance may be described as radiating from these bodies toward the peripheral gray matter, which latter is arranged in folds called convolutions or gyri. The cerebellum has an analogous structure, with variations—the corpus dentatum as central gray body, and white substance radiating thence to the peripheral convolutions. The pons Varolii is made up of white substance on its outer and anterior parts, with masses of gray matter within. In the medulla oblongata we find an analogous structure, white matter at the periphery and gray matter in the centre and posteriorly. The floor of the fourth ventricle contains a series of most important ganglia (masses of gray matter). The spinal cord has the same structure throughout its length—a structure best described upon a transverse section. Such a section is seen to consist of symmetrical halves, each containing a central gray mass and peripheral white matter. The gray matter is divided into anterior and posterior masses, the so-called horns, of which the anterior is the larger. The posterior horn reaches out to the margin of the organ, but everywhere else there is white substance outside of the gray horns. The shape of the gray horns and the relative proportion of white and gray substances vary in different parts of the cord, but that does not immediately concern us. From the spinal cord, symmetrical on either side, are the roots of nerves; the posterior roots being attached to the cord just inside of the posterior horn, the anterior roots issuing from the anterior mass of white matter. Upon the posterior root, just before it conjoins with the anterior root, we see a small swelling, which is a mass of gray matter—a ganglion. The white peripheral matter is usually subdivided in each half of the spinal cord into two parts—the antero-lateral columns, and the posterior columns—the dividing line between them being the posterior gray horn, where it strikes the periphery of the organ. A microscopic central canal runs the entire length of the spinal cord, continuous with the large openings called the ventricles, in the encephalic masses. Just at the junction of the spinal cord and medulla oblongata, in front, is seen a limited spot where bundles of white substance cross the median line, being myelinic fibres extending from the anterior column or pyramid of one side of the medulla to the antero-lateral column of the other side of the cord, constituting the so-called decussation of the pyramids.

The encephalic nerves all terminate in parts below the hemispheres and cerebellum. If I add that histologically and morphologically every part beneath the cerebrum, cerebellum, and opto-striate bodies belongs to the spinal cord, it will be truthful and of great help in our physiological and pathological studies. Ever since I began lecturing here, four years ago, I have taught this natural division of the nervous centres into the spinal axis, cerebellum, and cerebrum. Accepting this classification of the centres, we can also say that with the exception of the olfactory, all nerves are spinal, which philosophically is perfectly true.

I will now call your attention to certain great functions of the central nervous system—functions which involve the entering into activity of large tracts of nervous tissue extending over one or more of the organs just enumerated. I allude to the conduction of sensations, the transmission of motor impulses, the so-called reflex action, and co-ordination of movement.

In the first place, about the conduction of sensations. How are sensations formed in the spinal gray matter transmitted upward so as to be put within reach of the higher sensibility we call consciousness? To this question only a very partial answer is possible, mainly with reference to the direction of conduction. It is quite surely ascertained that a sensation originating in an irritation of the right lower extremity is perceived by the left cerebral hemisphere, and the left half of the pons Varolii. In other words, the sensory nerves, or better, the sensory paths, all cross the median line somewhere in the spinal axis. We owe chiefly to the experimental inquiries and pathological observations of Brown-Séquard the demonstration of two most important facts in this connection: (1) That these paths cross the median line (decussate) almost immediately after entering the cord, and then extend upward in the opposite half of the organ to the seat of consciousness; (2) and that it is the central gray matter, not the posterior columns, which contains these paths. This almost horizontal decussation has been shown to take place at the origin of every spinal nerve; in other words, sensory decussation is complete throughout the spinal axis. There seem be good experimental and pathological reasons for believing that the perception of sensations (their appreciation by consciousness) takes place in the pons Varolii; though cerebral action must intervene in the most complete perception, that including recognition of cause of irritation. I cannot leave the subject of transmission of sensations without adding a few words about a great law, a proper understanding of which is of great help to us in diagnosis—I mean the law of reference of sensations. By reference of sensations we mean the fact that when a sensory nerve is irritated at any point, at its termination (normal way), its middle, or at its origin in the spinal axis, the resulting sensation is felt in the parts to which the nerve is distributed. To illustrate: If I touch the table with my two outer fingers, I correctly refer the sensation to the vicinity of the pulp of these fingers; if I strike the ulnar nerve behind the elbow, the greatest sensation is felt in the tips of the little and ring fingers, which this nerve supplies, and if I could irritate the spinal origin of the ulnar nerve, or parts in physiological relation with it higher up, the sensation would still be felt in the district supplied by the nerve. You are all aware that persons who have lost limbs by amputation feel the absent member a good while; and they do so by virtue of this law. This law of reference of sensations holds good throughout the sensory tract (æsthesodic tract), from the special senses down.

In the second place, as regards the transmission of motor impulses from the nervous centres outward. Experiments and pathological observations have taught us that in the apparatus for motor manifestations (kinesodic tract) there is also a crossing over of paths. The motor fibres, or better, paths which transmit impulses to the right leg traverse the median line, though in a very different way from the sensory paths of the same limb. The right half of the spinal cord contains, throughout its length, the motor paths for the right limbs and right side of trunk; no decussation takes place until the lower edge of the medulla oblongata is reached, when all the motor paths cross the median line and enter the left side of the medulla to extend upward to the cerebrum. In the medulla and the pons Varolii the motor paths cross the median line rather higher up than the origin of the motor nerves. The importance of using the word paths instead of nerves, in the present state of our knowledge, is shown in this connection, since physiology and pathology teach that there is a crossing over, while anatomy seems to demonstrate that all motor nerves (anterior spinal roots) have their nucleus of origin in the corresponding half of the spinal cord; the right sciatic nerve springing from the right anterior horn of the cord, the right hypoglossal and facial nerves from nuclei in the floor of the fourth ventricle on the right side of the median line.

In the third place, the all-pervading function of reflex action—sensori-motor, excito-motor phenomena. The following definition of a reflex action is perhaps sufficient in a theoretical sense: it consists in the transformation, by nerve cells, of a sensitive impression (with or without consciousness) into motion, chemical action, or ideas. The parts essential to the performance of a reflex action consist of a centripetal (sensory) nerve to transmit the excitation, a ganglion cell to transform it into nervous force, and a centrifugal nerve to carry the nervous impulse to the muscle, gland, or cerebral convolutions. The results of the activity of such an apparatus are motion (common muscular, or vascular), secretion, ideation. From this definition you can readily imagine that reflex actions occur in nearly every part of the body, in small segments of it as well as in large portions. A heart cut out of the animal's body will continue to beat some time in response to irritations. Contractions may be obtained by irritating a small portion of intestine removed from the body, and a small segment of the spinal cord will suffice to give reflex movements in the muscles supplied by that piece of cord. Reflex actions take place in all parts of the nervous system (spinal axis, cerebrum, sympathetic system), and at all times; and it is through this kind of action that the most important bodily functions (including in part, certainly, cerebration) are produced. There is a tendency to make all active nervous phenomena of reflex nature, denying the existence of spontaneity in the animal frame; and I must admit that a good deal can be said in support of this extreme view. It would be quite out of place for me to enter into any details about individual reflex actions. I only ask you to remember that many mental manifestations are reflex in character; that respiration, circulation, nutrition, many acts of our life of relation (walking, etc.), are under the control of the law of reflex action; and that many diseases are produced by just the same mechanism.

Lastly, concerning co-ordination of movements. It has been thought, and that within twenty-five years, that there existed a "faculty" of co-ordination (what "faculties" have not been invented by fertile brains!) by the exercise of which our movements are regulated and made perfect. Such a view I need hardly tell you is quite opposed to present physiological and psychological notions. Simple experiments performed long ago show the absurdity of this creation of the theorizer. If the cerebellum or cerebrum be removed from an animal, it is noticed that in the animal deprived of cerebrum there is no impairment of co-ordination at any time: in the case of injury to the cerebellum inco-ordination occurs, and lasts for quite a while; but as shown by Dalton, Lussana, and Weir Mitchell, this disorder ultimately ceases. I have already referred to the experiment of putting nitric acid upon a frog's anus after decapitation, with the effect of causing perfectly co-ordinate movements resulting in the removal of the acid; which movements are so perfect as to have led one German physician at least to admit a consciousness and volition of the spinal cord. These experiments show that co-ordination is a function of many parts of the nervous centres. Another way of studying this function is by watching the physical education of an infant. At first his motions are utterly purposeless and inco-ordinate, but by degrees he acquires the power of moving groups of muscles in a definite way, and at length comes to prehend, to stand, to walk, to speak; and when older he may learn to eat, to play on the piano, to do astonishing feats of hand-skill. By analyzing this progress, we reach the conclusion that parts of the nervous centres are educated by repetitions of sensory impressions and of volitional motor impulses, leaving their impress upon groups of cells (so-called centres) which have the property I propose to call retentivity: the cells acquire the "habit" of acting in an automatic, necessary way. It is, furthermore, important to notice in this connection the fact that it is impossible for us to move one muscle alone by a volitional impulse; in other words, the simplest act is co-ordinate. For example, the external rectus muscle of the right eye cannot be made to contract without consentaneous contraction of the internal rectus of the opposite eye; in willed flexion of the hand the extensor muscles contract as well as the flexors. The best theory of co-ordination, to my mind, is that which, denying all direct (continuous) connection between the cerebrum and individual muscles, admits the existence of educable or educated groups of motor cells in all parts of the spinal axis, which groups act as wholes upon the reception of a volitional impulse. We will to grasp a pen, and, after having learned, we do it without giving any attention to the details of the movements. The necessity of watching movements which should be performed in an automatic way is a serious symptom of disease.

To resume, there are four generalized functions in the nervous system.

1st. Sensation and perception are executed by means of paths which decussate almost horizontally in the spinal axis; the conduction being by the gray matter, not by the white columns of the cord; coarse sensibility with doubtful consciousness has its seat in the pons Varolii; perfect perception and appreciation is possible only with the help of the cerebral mass.

2d. Motion is executed through motor impulses, which, starting from the opto-striate bodies (from cortex of cerebrum also?) traverse paths which decussate almost opposite the motor nerves as far down as the lower margin of the medulla oblongata, where the paths for the trunk and limbs decussate in a bundle, to remain below this point in that half of the spinal cord whence arise the nerves going to the muscles.

3d. Reflex action is the result of a transformation of an irritation from the periphery into nervous force by a nerve cell, transmitted centrifugally by a second nerve. That all nervous phenomena are of reflex mechanism is not to be too positively denied.

4th. Co-ordination is no faculty, but a function of every portion of the motor tract of the spinal axis from the origin of the third cerebral nerve down.

There is not time for, nor had I the intention of, entering into an analysis of the restricted functions of the organs composing the nervous centres. Even Hitzig's and Ferrier's most interesting researches into the possible motor functions of the cerebral convolutions I must pass by, intending to speak of them in the course of remarks upon the cases which we shall study together this winter.

There are a few pathological laws logically allied to the physiological propositions enumerated above, which I want to submit to you.

1st. Any disease of any part of the nervous centres may produce two kinds of symptoms, which we should always attempt to distinguish: these being symptoms of irritation, consisting, according to the location of the lesion, in exaltation of ideas, delirium, in numbness, pain, and in spamodic movements; and symptoms of destruction of parts, loss of mental power, anæsthesia, paralysis. Brown-Séquard was, I believe, the first to insist upon the exceeding importance of distinguishing these two classes of effects.

2d. It should be borne in mind that irritating lesions may cause the second class of symptoms by producing an inhibitory (arresting) effect upon centres near or distant.

3d. Ischæmia of the nervous centres produces extreme irritation symptoms, delirium, spasms, pain, and numbness, followed by loss of function of parts.

4th. The effects of hyperæmia are not satisfactorily known.

5th. A want of equilibrium in the circulation of both hemispheres is a common cause of vertigo.

6th. Almost any lesion of the nervous centres may disturb the nutrition of distant (non-nervous) tissues.

7th. A generalized lesion of the convolutions of the brain produces, first, exaltation of mind and emotions, followed by abolition of the faculties, and a false general paralysis.

8th. A lesion of one cerebral hemisphere gives rise to symptoms (paralysis, numbness) in the opposite side of the body and face. The localization of the lesion in the left hemisphere about the fissure of Sylvius, is exceedingly likely to abolish language, spoken and written; while lesions of the right hemisphere produce more severe palsy, set the emotions free, and endanger life more.

9th. A lesion of the centre of the pons Varolii will produce general paralysis, with probably anæsthesia and changes in the bottom of the eyes.

10th. A lesion in one-half of the pons Varolii will produce palsy with (probably) anæsthesia in the opposite side of the body.

11th. Lesions of the cerebellum when in one lobe produce an incomplete hemiplegia on the opposite side, with marked eye and stomach symptoms.

12th. A suddenly produced lesion of the centre of the medulla oblongata will probably kill the patient at once by arresting the respiration.

13th. A lesion localized in one-half of the medulla oblongata will give rise to hemiplegia and anæsthesia on the opposite side.

14th. A lesion at the base of the brain, not on the median line, will produce a crossed palsy (as first indicated by Romberg); palsy of body on side opposite lesion, and palsy of one or more cranial nerves on the same side as the disease.

15th. Pressure anywhere within the skull may affect the nutrition of the optic nerves.

16th. In lesions of the cerebral hemispheres accompanied by coma (apoplexy), the eyes are together turned and fixed toward the side of the lesion, and away from the palsied side.

17th. A lesion occupying the whole thickness of the spinal cord, or its gray matter, will give rise to palsy of all parts below the lesion, *i.e.*, below the distribution of nerves issuing from just above the lesion; and such a paraplegia is necessarily attended by anæsthesia, and increased reflex movements in palsied parts.

18th. A lesion in one-half of the spinal cord (hemisection, Brown-Séquard) at any point will produce paralysis with hyperæsthesia on the same side as the lesion, and anæsthesia on the opposite side.

19th. A lesion involving the posterior columns of the spinal cord produces neuralgia and ataxia of movements.

20th. A lesion affecting the lateral columns of the spinal cord will cause a paralysis accompanied by contracture.

21st. A lesion of the cells of the anterior horns of the cord alone will produce a palsy (no anæsthesia), accompanied by extreme wasting of muscles, and loss of electro-muscular reaction. Any part of the spinal axis may be the seat of this disease.

22d. A lesion (destructive) of nerve trunks gives rise to a paralysis with anæsthesia, and rapid loss of electro-muscular reaction.

23d. A lesion in the cerebrum and the opto-striate bodies may produce secondary lesions in the spinal cord and nerves.

24th. A lesion of the spinal cord may cause secondary lesions upward and downward in the cord, and in nerves.

25th. Lesions of nerve trunks may produce secondary lesions of the spinal cord.

I.—ON HYSTERICAL SYMPTOMS IN ORGANIC NERVOUS AFFECTIONS.*

I PURPOSE in this paper calling attention to some points in the semeiology of diseases of the nervous system which have as yet been little studied—perhaps not at all in this country. While I do not know that any author has written upon this subject, I wish to say that I was first led to observe the emotional state of paralytics through a remark of Prof. Charcot, of Paris, made either at the *Société de Biologie*, or in his wards in the Salpêtrière Hospital, during the winter of 1869–70. I have since that time frequently spoken of hysteroid symptoms as occurring in certain paralytics, in lectures, and have called the attention of the resident staff at the Epileptic and Paralytic Hospital on Blackwell's Island, New York, to the matter. I am also aware that Dr. Brown-Séquard has observed and studied such symptoms in private practice. I should also say that a recent writer on the pathological physiology of the cerebrum,† has ably written of symptoms closely akin to those I shall describe, and has (after Brown-Séquard) pointed out how different are the symptoms produced by lesions of either cerebral hemisphere. I ought not to omit a reference to a remarkable paper, written a good while ago,‡ in which pointed attention is paid to the emotional state of hemiplegic patients, whose intellectual condition is criticised by the author, Dr. B. W. McCready.

The term "hysterical symptoms" is one which seems useful for purposes of clinical study and classification, and one, consequently, to be retained until we shall have exactly analyzed and re-classified the signs which, together, go to form the classical hysterical state, or hysteria.

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* From the Archives of Electrology and Neurology for May, 1875. This paper was read before the N. Y. Society of Neurology and Electrology at the meeting for June, 1874.

† De Fleury; Du dynamisme comparé des hémisphères cérébraux. Paris, 1873.

‡ To what degree are the Intellectual Faculties affected in cases of Apoplexy and Hemiplegia? N. Y. *Journal of Medicine*, new series, vol. iii., p. 203 (1857).

Before proceeding to relate the cases which form the basis of this essay, I may be pardoned for presenting in tabular form an analysis of the chief hysterical symptoms.

- HYSTERICAL SYMPTOMS.
  - Intellectual..
    - Simple eccentricity of conduct.
    - Impairment of logical capacity.
    - General ideational disorder (mania).
    - Concentration of attention upon idea or sensation (ecstasy).
  - Emotional...
    - Psychic pain.
    - Crying, Laughter — with co-incident spasms and secretions.
    - Morbid impulses.
  - Sensorial....
    - Hyperæsthesia..
      - Special senses.
      - Ovary (left).
      - Infra-mammary region (left).
      - Vertebral groove (left).
      - Whole surface of body.
    - Anæsthesia.....
      - Special senses (hemiopia, amaurosis, deafness).
      - Skin......
        - Hemiplegic (left).
        - Paraplegic.
        - Universal.
      - Deep parts.
        - Loss of muscular sense.
        - Retention of excretions.
    - Dysæsthesia....
      - Pain......
        - Side of head (left) (clavus).
        - Left breast.
        - Various neuralgiæ.
      - Ovarian aura.
      - Globus hystericus.
      - Numbness.
      - Sense of heat or cold.
      - Hallucinations.
  - Motorial.....
    - Hyperkinesis...
      - Tetanoid seizures.
      - Hystero-epilepsy.
      - Local contractures.
      - Postural spasms.
      - Catalepsy.
      - Cough and vomiting.
    - Akinesis.......
      - Paraplegia.
      - Hemiplegia (left) (not facial).
      - Retention of excretions; incontinence.
  - Secretory....
    - Excess of tears.
    - " " urine.
    - " " intestinal gas.
    - Suppression of urine.
    - Local congestion and ischæmia.
  - Imitation of various organic diseases (arthritis, etc.)

Philosophically considered there is nothing specific in hysterical symptoms; they are functional disturbances of various organs, due to a morbid dynamical state of the nervous system. With this understanding, it is not difficult to conceive of the very large number of symptoms which may receive, and justly in a certain sense, the qualification hysterical. The term hysteria, and the adjective derived from it, I shall make use of without having much respect for it; it was imposed upon nosologists by the dominant theory of olden time respecting the pathological physiology of the morbid state in question; a theory holding that the uterus and its appendages were the seat of the morbid process. With the various theories of hysteria, their rise and fall, and rehabilitation, I have only this to do: to state that most physicians to-day believe that the central nervous system is at fault, dynamically, in hysteria. Some writers teach that the spinal cord is most disordered in this condition; others, that the organ whose badly performed functions are exhibited by "hysterical symptoms" is the cerebrum.

The following cases are offered as illustrations of the proposition that hysterical symptoms will present themselves in persons suffering from organic disease of the nervous system. A natural division of the cases is adopted, into cases of disease of the brain, and cases of disease of the spinal cord. Cases observed by myself are marked by a prefixed asterisk.

CLASS A.

Cases of Disease of the Spinal Cord.

*I.—Female, E. E., 36 years, born in New York. Left hemiplegia, with paresis of right limbs—well marked hysterical symptoms; suppuration of left elbow joint; death, extensive central myelitis, with formation of cavities in cord.

This patient was admitted to Epileptic and Paralytic Hospital, Sept. 17th, 1868. Five years before had sudden right hemiplegia, cured in three months. About three years ago, while under strong depressing emotion, sat out of doors three days and two nights, when palsy of left lower limb was found. Two months before admission had pins-and-needles feeling in left arm, and gradually lost use of it. On reception, patient *nervous;* has left hemiplegia, (paresis of arm), with contractured leg in flexion; palsied parts are seat of prickings, and are cooler than right limbs; cannot retain urine; speech (articulation) impaired. August 23d, 1871: has double rotatory nystagamus; no diplopia, pupils normal; no facial palsy; left hemiplegia with contracture of fingers (slight), of knee and foot. Complete anæsthesia of left arm, slight (with numbness) of lower limb; right arm only weak; incontinence of urine.

This patient exhibited almost constantly, during the several months I observed her, well-marked hysterical symptoms. She had a squeaky, tremulous voice, which changed easily into a natural tone when she was chided. Often she began to cry, flush, and shed tears on speaking to me, but if I passed on she would control herself. I may state that the larynx was normal. I often hesitated in my diagnosis because of the coincidence of these emotional symptoms with left hemiplegia. She died on February 16th, 1873, in consequence of exhaustion from the elbow-joint lesion, and a sacral bed-sore. The spinal lesion has been spoken of, and it remains only to add that no lesion was found in the cerebral hemispheres.

*II.—Female, æt. 32; single. Admitted to Presbyterian Hospital, November 16th, 1872. Attacks of gastric pain and vomiting; fulgurating pains in extremities; palsy of left 3d nerve; locomotor ataxia. Sudden death; cerebellar hemorrhage; sclerosis of posterior columns of spinal cord.

Many details of this case are interesting for the student of locomotor ataxia, but need not be reproduced here. Suffice it to say that this patient was remarkably emotional and hysterical in manner, so much so that hysteria was thought to be her only disease, manifesting itself in emotion, vomiting, numbness of left arm, and neuralgia. The nurses and the resident staff could not be fully persuaded that the patient had organic disease. Upon examining her on taking the service, January 4th, 1873, I became convinced, from the coexistence of fulgurating pains, ataxia in upper and lower extremities, and left 3d nerve palsy, that the case was one of sclerosis of the posterior columns, complicated by hysteria. Her death took place as follows, according to the hospital case-book: "April 6th, again hysterical; complains of left arm. April 14th, was taken with severe hysterical (?) convulsions at 3.30 P.M., which were quite continuous and lasted about an hour. There was no vomiting, and nothing to show pure hysteria, such as she had had before. When the convulsions ceased patient fell into a sleep, as was generally the case after the attacks of hysteria. In this sleep there was nothing remarkable, and about 7 P.M. the nurse, having her attention called to her by hearing a loud sigh, found her in apparent syncope. On the arrival of the resident physician the patient was dead."

*III.—Female, æt. 23; single. Sclerosis of cord; hysterical symptoms. Seen October 20th, 1873. A nervous girl, with occasional irregularity of menstruation, but no dysmenorrhœa; at times hysterical laughter or tears; never convulsive attack. In July, 1871, while out walking, after having climbed a number of walls, felt weak and awkward in right leg; thought she had sprained her knee. There is not enough evidence to support this statement. Ever since she has had weak right leg, without anæsthesia or numbness; at times more use of leg than at others; almost cured once or twice; of late has required help of crutch or friend's arm in walking. When I examined Miss DeP. I found paresis of the right leg, the loss of power being marked at ankle and toes; there was doubtful weakness of the right hand; I could not make out that the knee-joint was affected. The muscles of the right leg showed a slight diminution of reaction to Faradic current, and this agent also showed that sensibility to pain was a little dull in leg and foot. In view of the history of the case, the capricious development of the palsy, the absence of reliable signs of central disease, the presence of a strong neurotic element in the family, and the fact that strong emotions had been acting upon her, I concluded that the patient had a functional palsy of an hysterical nature. Strychnia was given her and Faradism used. The specific effects of strychnia appeared, and the patient was decidedly tetanized for a while; this passed off, and when I last saw the patient, on December 11th, she was in about the same state as at the beginning of treatment. The unfavorable effects of the treatment led me then to believe that the patient had an obscure central lesion, probably sclerosis.

In March or April, 1874, patient rapidly grew worse, becoming paraplegic and her hands showing paresis. In July she was placed in an irregular water-cure house, where extensive bed-sores formed, in consequence of want of care and of cold applications to the palsied parts. (She had *continuous* applications for several days.) Exhaustion and pyæmia caused death, August 1st. The post-mortem examination showed disseminated sclerosis of the spinal cord. The brain not examined. I have the specimen in fluid, and will make a detailed report of the lesion. Dr. Chas. A. Leale, of this city, treated the patient during July, after the bed-sores had formed, and I made the autopsy at his request and that of deputy coroner Dr. Shine.

IV.—Case by Duchenne. In a female who had true hysterical paraplegia, in Trousseau's service. Dr. Duchenne discovered that patient was suffering from the characteristic fulgurating pains of locomotor ataxia, and had noticed them five years. Dr. Duchenne diagnosed the coexistence of hysteria and locomotor ataxia, and foretold that after the cure of the paraplegia the ataxic movements would reappear. This did happen after the use of Faradization.

Electrisation localisée, p. 653, ed. 1872.

V.—Female, æt. —. Hysteria and sclerosis of the lateral columns of the spinal cord. The patient began to have attacks of convulsive hysteria about the age of 14 years, and occasionally thereafter; at 34, after such an attack, had a left hemiplegic contracture, which lasted a fortnight and suddenly disappeared. The next year there was a second attack of contracture, at first hemiplegic, and then bilateral. After two years patient improved so much as to be able to walk about; then a relapse occurred after an hysterical attack. Death by an intermittent disease. Post-mortem examination by Bouchard showed sclerosis of both lateral columns of the spinal cord from medulla downward. The early history of case was taken by Briquet in 1850, and completed by Charcot at the Salpêtrière.

Charcot. Soc. méd. des hôpitaux, séance du 25 janvier, 1865; in Gaz. hebdom., 1865, p. 109.

VI.—Female, æt. 36. There were symptoms of sclerosis of various parts of the spinal cord and medulla oblongata; hysterical attack. At time of report patient still alive. She was the sister of two females affected with disseminated sclerosis of nervous centres. From an early period she had had attacks of convulsive hysteria, throwing herself about in bed, uttering monotonous cries; the respiration reduced; apathy and seeming unconsciousness closing the scene. These attacks were usually brought on by emotional disturbances.

Friedreich: Ueber degenerative Atrophie der spinalen Hinterstränge. Virchow's Archiv, 1863, Bd. xxvi., p. 391 et seq. Bd. xxvii., p. 1. (Case of Lisette Süss.)

In the above six cases of organic disease of the spinal cord the following hysterical symptoms were observed and noted:

Abnormal emotional tendency in all cases.
General nervousness in cases I., II., III.
Tears and sobs upon slight provocation in case I.
Tremulous variable phonation in case I.
Disordered sensibility on the left side in cases I., II.
Disordered sensibility in lower limbs in case IV.
Vomiting in case II.
Temporary paralysis in case IV.
Contracture of limbs in case V.
Convulsive attacks in cases II., V., VI.

The commingling of these hysterical symptoms with the signs of organic disease has caused each case to present a peculiar problem to the examining physician. Sometimes the organic disease was wholly overlooked. In Trousseau's and Duchenne's case (IV.) the diagnosis of hysterical paraplegia had been correctly made, and the sclerosis of the posterior columns of the cord not suspected by the former celebrated clinician. Charcot's case (V.) is still* referred to by him as one of hysteria, in which a lesion was found—a view which I would suggest is the inversion of the correct one. As regards my own cases, in Nos. I. and II., there were times when I was in much doubt as to whether all the phenomena were not functional. It was not until alterations of nutrition appeared in Eagles (I.) that I became firm in my conviction that there was a spinal lesion. Her general appearance, manner, and speech, and the existence of many of her symptoms upon the left side, made up a more strongly marked picture of hysteria than I can give any idea of by words. In Miss L.'s case (II.) my faith in the significance of fulgurating pains and coexistent 3d nerve palsy kept me right. Even after I had made the diagnosis of sclerosis in this patient, the impression of the medical gentlemen who saw her was that she was simply hysterical. In the case which I have added since reading this essay, Miss De P. (III.) I made a grave mistake in diagnosis. I am glad to be able to publish this case as a guide for other physicians.

From a study of these few cases, in the present state of our knowledge of pathological physiology, it seems impossible to point out any close genetic relations between the lesions found and the hysterical symptoms observed. In support of the view that there was merely a coincidence in these six cases, I would adduce the following considerations: *a*. That the organic diseases were various. Two patients had sclerosis of the posterior columns of the cord; two (including case III. not yet minutely studied) disseminated sclerosis; one sclerosis of the lateral columns; and one extensive central myelitis. *b*. That the number of cases of disease of the spinal cord in the books and periodicals I have been reading in several years is quite large, and that these six cases form an insignificant minority. *c*. A certain number of cases of fatal hysteria have been examined after death, without any lesion of the central nervous system being discovered.*

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* Charcot: Leçons sur les maladies du système nerveux, Paris, 1872-3, p. 316, 17.

After this conclusion of coincidence, I can only call the attention of this society to one question connected with these cases, viz., that of their bearing upon diagnosis. There are reasons for believing that hysterical persons are sometimes treated for organic diseases of the spinal cord, which exist only in the physician's mind; and the cases I have related show how possible the converse error is when the hysterical symptoms are so prominent as to prejudice the physician; and we may thus be led to attempt severe and unsuccessful treatment, and to make a false prognosis.

The only way in which we can hope to avoid these errors is by having a clear understanding of what symptoms are hysterical, either essentially so or by association in groups; and by being prepared to appreciate and firmly believe in the true meaning of cardinal symptoms of organic disease of the spinal cord; as the fulgurating pains and 3d nerve palsy of posterior spinal sclerosis, the paresis, ataxia, and peculiar speech of disseminated nodular sclerosis, the alterations of nutrition, and the abnormal muscular reactions to electricity in myelitis, etc. I believe that in minute and exact analysis of symptoms and symptom-groups lies our only safety.

CLASS B.

Cases of Disease of the Brain.

*I.—Mrs. R., 75. The subject of extensive arterial degeneration. On September 16th, 1873, a left hemiplegia was quickly developed without loss of C. There was complete anæsthesia also on left side of body. In the course of three weeks there was no improvement in palsy, or anæsthesia, contracture appearing, and patient became very much depressed and careless of result ; this melancholy being in striking contrast with her previous condition. No improvement during winter. The general health remained remarkably good ; the heart and arteries showing signs of progressing disease. No fever. The most striking symptoms were of an hysterical nature ; the patient crying like a child for a moment, without any sufficient provocation. Upon the physician saying "Good morning," this lady would burst into convulsive crying, with enormous facial contortions—no tears, but moaning just like a child. Suddenly the fit would pass off, the face become natural in an instant, and the patient exclaim, in a provoked way, "Oh, what an old fool I am !" During the short medical visit, in a few minutes, several such paroxysms would occur. Besides, in the last few months of life there were great physical restlessness, peevishness, loss of memory, hallucinations (eye and ear), and delusions. Death toward end of May, 1874 ; no autopsy allowed. It should be added that this lady had possessed remarkable intellectual power, and unusual force of will; that during the winter, often immediately after crying attacks, she made very witty remarks. This lady was a patient of Dr. Wm. H. Draper, with whom I saw her.

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* L. Meyer: Ueber acute tödliche Hysterie. Virchow's Archiv, 1856, p. 98, Bd. ix.

*II.—Midwife, æt. 46. Left hemiplegia. Some prodromata in the shape of vertigo last summer, and much headache. Sudden palsy of left side without loss of C. When examined at the Epileptic and Paralytic Hospital, Blackwell's Island, on January 30th, 1874: presents a complete left-sided palsy, with marked anæsthesia of left face ; great loss of sensibility in left arm and leg; does not know where arm lies ; has beginning secondary contracture. The intercostal muscles on the left side are much palsied; the heart shows a faint apex systolic murmur; no gout or syphilis. In the middle of February marked hallucination of sight and hearing occur. Left (palsied) palm 1.4° C. warmer than right. In March, April, May, and since, patient is often very emotional, though perfectly rational. Bursts into tears with much facial contortion, saying she does not know why. Some improvement in leg; none in arm, though sensibility is everywhere much better.

*III.—Male, æt. 28. Left hemiplegia, from probable embolism of a cerebral artery. Came to Clinic for Diseases of the Nervous System at the College of Physicians and Surgeons, where it was learned that eight weeks before, when going to bed, he had a slight vertigo, with tingling in left side of tongue, and numbness, followed by loss of power in left limbs. No loss of C. Was in bed three weeks, because of inability to walk. Has since improved steadily. Examination shows a common left-sided hemiplegia, with involvement of face and tongue. There is a basal systolic heart-murmur. On June 13th, 1874, very much improvement is noted. During examination the patient flushes. He states that since attack he has been unduly emotional; when annoyed he has felt something rising from chest into throat, preventing speech; has even wept a few times.

*IV.—Female, 28 ; married. General paresis, hysterical symptoms. In June, 1873, began to complain of severe headache, universal, more severe on right side; not nocturnal, occurring in non-periodic paroxysms. Pain down the spine. In July gradual failure of sight; some loss of memory; using wrong words. Generalized weakness first noticed by friends during December. No delirium. Since June occasional trembling of hands. From the first has had feeling of pins and needles in all her limbs at extremities. Has also had attacks of suffocation—something filling up her throat. Much constipation and nausea. In last two months less headache. Examination on November 11th, 1874 : patient so weak as to be hardly able to stand; totters much and inclines a little to left side; speaks loudly and complains of darkness of room. Speech clearly articulated and rational. Memory much impaired; hands very weak; no facial palsy or muscular atrophy. Ophthalmoscope shows retinal vessels issuing from a uniformly red and velvety ground; choked disks. No anæsthesia. Very deaf on left side. Has been very emotional at times, and now presents an hysterical manner. Has been seen several times by Brown-Séquard, who thinks that she has a brain tumor.

*V.—Male, æt. 18. Left hemi-chorea with paresis, 3d pair palsy on right side; probable lesion of right crus cerebri; hysterical laughter. This young man had paresis of left arm gradually developed from May, 1873. In July the choreic movements first appeared. On December 6th, left arm, leg and face are seat of rather ataxiform choreic movements; no palsy of eye muscles. Toward middle of January, 1874, hebetude, greater chorea, slight ptosis on right side. During late winter and spring progress in paresis and 3d pair palsy ; hebetude, but no loss of memory. Often laughs without cause, and finds great difficulty in stopping the laughter; more correctly, patient has, involuntarily, the special spasm of full laughter very often, without the psychical elements. August, 1874, complete 3d pair palsy, with left hemiplegia and chorea; at times some spasm in right arm; intelligence good. Patient died in September, but no autopsy could be obtained.

VI.—Male, æt. 43. Left hemiplegia. When twenty-two years of age had a chancre followed by secondary manifestations. Twelve years ago had an attack of hemiplegia, involving left side of face, and left limbs. The paralysis came on slowly ; soon after a midday dinner he noticed that the leg was weak. In the course of an hour this became entirely paralyzed, and the arm became enfeebled. At first, some improvement, none in last few years. Examination (spring of 1874) shows a left hemiplegia without contracture, except in face, where some muscles show some slight clonic spasms. Since a short time after the attack, has been easily excited, either to laughter or tears; on which account he has been unable to go to church. Mind clear; some impairment of memory. Has atrophy of right optic disk.

Observation by Dr. T. A. McBride, of New York.

VII.—Male, æt. 39. Hysteria; left hemiplegia; semi-coma; thrombosis of basilar artery. Patient had been weak and stupid for two years. A fortnight before admission had vertigo and repeated attacks of a hysterical nature, with sobs, and bursts of laughter. Five days before death developed left hemiplegia, and passed into comatose state.

John W. Ogle, in Trans. of Path. Soc. of London, 1864, p. 14.

VIII.—Male, æt. 59. Left hemiplegia; speech preserved; much anæsthesia of palsied limbs; impairment of sight and hearing on left side. In three months some periodical delusions. Is at times given to laughing in an almost insane way; after which there supervenes a strong tendency to weep. Death in eleven months.

De Fleury: Du dynamisme comparé des hémisphères cérébraux. Paris, 1873. Pp. 12, 34.

IX.—Female, æt. 74. Left hemiplegia; speech preserved; various alterations of sensibility (anæsthesia of left limbs, ear, and eye). When asked to perform movements patient cries and sheds tears. Later hallucinations and delusions. Death in nine months, with palsy of right side.

De Fleury: Op. cit., pp. 124, 5, obs. xii.

X.—Male, æt. 42. Right hemiplegia, with aphasia. Subsequently frequent epileptic convulsions, which diminished in frequency later. Intellect remained clear; partial recovery of limbs. "In the early years of his infirmities crying fits would often occur, especially when meeting an old friend, and no one near him to interpret, or when he would see the promotion of a classmate, or on recovering from an epileptic attack, the tears would flow in torrents. . . . . . . On the other hand, a jest, an anecdote, or frolic of any kind would excite such convulsive laughter that I have again and again feared for his life, from the evident determination to his head. . . . . . . While engaged in any matter of interest his breathing becomes almost stertorous; his salivation is profuse." . . . . . .

Dr. B. W. McCready: To what degree are the intellectual faculties affected in cases of Apoplexy and Hemiplegia? N. Y. *Journal of Medicine*, iii., Sept., 1857, p. 203.

XI.—Male, æt. 60. Right hemiplegia and aphasia; intellect good. "The patient Wilcox weeps as often as the physician calls attention to his misfortune. His face becomes as much distorted as that of a weeping child, and his tears flow freely."

Dr. McCready: Op. cit., p. 221, and p. 246.

XII.—Male, æt. —. Double hemiplegia. Left side palsied first. While paralyzed in left side only, and still able to speak (on first day), he was as sound in mind as ever in his life; yet he wept frequently, with a child's distortion of face.

Case by Prof. A. Clark in McCready, op. cit., p. 236.

XIII.—Male, æt. 58. General paresis—greater on left side. Symptoms of organic cerebral disease, general paresis, epilepsy, speech much affected, writing scarcely legible, intellect clear. "He does not weep, but laughs immoderately on every trifling occasion. He scarcely smiles, but is seized with convulsive, hysterical laughter." . . . . . . "He takes frequent and convulsive inspirations preparatory to uttering his words." No autopsy.

Prof. A. Clark, in op. cit., p. 246.

XIV.—Female, æt. 52. Partial left hemiplegia, right hemiplegia of two years' standing; palsied limbs rigid; absolute incapability of articulating sounds. "Every effort of the patient ends only in unintelligible stammering, interrupted by plentiful tears and sobs. Tears and sobs, such are the only means of expression in her power, and she uses them largely, for it is enough to feel her pulse or to speak to her to provoke an abundant flow of tears, a purple color of the face, and convulsive action of the muscles of respiration;

deglutition is embarrassed, and the patient makes us understand by gestures with her left hand, that the pharynx acts with difficulty." Intellect clear. Autopsy showed softening of the pons Varolii.

CRUVEILHIER, in Anat. pathol., liv. xxi., p. 3.

XV.—Female, æt. 55. Four attacks of left and right hemiplegia, with great difficulty of articulation; understanding preserved. She weeps whenever questioned. The autopsy showed a clot in the left hemisphere and cicatrices in the right hemisphere and in the cerebellum.

CRUVEILHIER, op. cit., liv. xxxviii., p. 1.

XVI.—Female, æt. 60. Left hemiplegia. An apoplectic attack in a subject of supra-orbital migraine. Although medical history is quite full, hysteria is not mentioned as having been present in earlier life. At beginning of attack had, with paresis of left limbs, "*une succession sans motifs de pleurs et de ris.*" Death in twelve days after beginning of palsy, which, at the last, involved also right limbs. Autopsy showed softening of corpora striata, and of centre of pons Varolii.

CRUVEILHIER, in LALLEMAND: Recherches sur l'encéphale, t. i., p. 101. Paris, 1824.

Of the above sixteen subjects:

Nine were males (III., V., VI., VII., VIII., X., XI., XII., XIII.).

Seven were females (I., II., IV., IX., XIV., XV., XVI.).

The paralysis was distributed as follows:

Right hemiplegia, two cases (X., XI.).

Left hemiplegia, nine cases (I., II., III., V., VI., VII., VIII., IX., XVI.).

Double hemiplegia, five cases (IV., XII., XIII., XIV., XV.).

Consequently fourteen out of sixteen patients had hemiplegia on the left side.

The hysterical symptoms present were not very various.

Undue emotions (tears and sobs) in fifteen cases.

Irrepressible laughter in five cases (V., VI., VII., VIII., X.).

Anæsthesia of left side of body in four cases (I., II., VIII., IX.).

Globus hystericus in two cases (III., IV.).

The lesions of the brain were determined by post-mortem examination only in a few instances.

The following table represents the pathological diagnosis:

Hemorrhage in both hemispheres, one case (XV.).
Unknown " " two cases (XII., XIII.).
Probable embolism of right cerebral artery, one case (III.).
" thrombosis of " " one case (VI.).
Unknown in left hemisphere, two cases (X., XI.).
" right " four cases (I., II., VIII., IX.).

Thrombosis of basilar artery, one (VII.).
Softening of pons Varolii, one case (XIV.).
" pons and corpora striata, one case (XVI.).
Probable cerebral tumor, one case (IV.).
Disease of right crus cerebri, one case (V.).

Emotional symptoms were present in fourteen cases of left hemiplegia, and in two cases of right hemiplegia. This disproportion is enormous, considering that the records of many authors have been diligently searched for cases. I think that we may conclude that patients with right hemiplegia (who so often lose their speech), hardly ever lose control over their emotions; while the subjects of left hemiplegia often do. Right hemiplegics are comparatively cheerful; left hemiplegics are depressed and prone to weep. I am led by my recent experience to believe that this law will be brought into much greater prominence by future statistics.

The question of difficulty in diagnosis which occupied us while discussing the spinal cases, seems to me of minor importance in cerebral cases. Two of the sixteen cases are worthy of remark in this connection. In one instance (case XVI.) the paralytic attack was preceded by a well marked fit of hysterical weeping; in another instance (case IV.) the exact diagnosis remained uncertain until the existence of organic brain disease (tumor?) was made sure by finding well-defined choked optic disks.

The thoughts which have arisen in my mind in connection with these relate to three points.

a. The possible parallelism between cases of hemiplegia from organic brain disease accompanied by hysterical symptoms, and typical hysteria.

b. The pathological physiology of some of the symptoms studied above, such as loss of control over the emotions, hemiplegia, and hemi-anæsthesia.

c. The new question of difference between the two cerebral hemispheres, in their functions and morbid susceptibility.

a. I have already, at the beginning of this essay, laid the foundations for comparing hysteria and the effects of certain cerebral diseases.

1. In typical hysteria the emotional symptoms are the most common, and according to many authors the most characteristic.

In all the cases of cerebral disease above related there were undue emotional manifestations, or emotional movements not duly controlled.

2. In typical hysteria many of the objective phenomena are almost always shown on the left side of the body; and we may consequently feel sure that in these cases the right hemisphere is disordered.

In nearly all of the above sixteen cases the right hemisphere was the seat of organic disease, and the symptoms were upon the left side of the body.

b. The genesis of symptoms in cases of organic disease and of functional hysteric disturbance. Adopting as I do, with some reservations, Brown-Séquard's new hypothesis, that cerebral lesions produce the symptoms which point out their existence, not by *destroying organs* in the brain, but by setting up irritations which *arrest* (inhibit) the functions of other parts of the encephalon, I find no difficulty in understanding why the same symptoms may exist without as well as with a brain lesion. In typical hysteria the functions of parts of the encephalon included in the right hemisphere, or in physiological relation with it, are inhibited by a peripheral irritation, starting from a diseased or disordered sexual apparatus, or other part; and, in case of organic cerebral disease, the same inhibitory action is produced. In both kinds of cases we may have loss of rational control over the emotions, loss of voluntary power over one half of the body, and loss of sensibility in the same part.

In cases of hemi-anæsthesia due to lesions in the neighborhood of the thalami optici (Türck,* Charcot †), the explanation is, I think, the same—that a lesion in this particular locality is more likely to inhibit the functions of the (sub-cerebral?) centres for perception of sensitive impressions than lesions of any other part. I have long believed with Brown-Séquard, that it is just so in the case of aphasia: We are forced by cases to deny the existence of an organ of speech in any convolution, yet are equally obliged by statistics to admit that a lesion of the posterior part of the left third frontal convolution, and immediately subjacent parts, is much more certain to inhibit the complex cerebral functions which co-operate to form articulate language, than any other cerebral lesion.

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* Sitzung der K. K. Akad. der Wissenschaften zu Wien, 1859.

† Op. cit., p. 271 et seq.

c. I may be pardoned for adding a short review of what seems well established concerning the different results of lesions of either cerebral hemisphere. After the great advance caused by the numerous publications upon aphasia, Brown-Séquard pursued the inquiry. In 1870[*] he communicated his conclusions to the Biological Society of Paris. He found that after lesions of either hemisphere the following symptoms predominated.

Left Hemisphere.	*Right Hemisphere.*
	Eschars.
	Œdema.
	Palsy of sphincters.
Aphasia.	More fever.
Palsy of organs of articulation.	Greater mortality.
	Pulmonary congestions.
	More frequent deviations of eyeballs in coma.
	Greater palsy.

The last three characters of left hemiplegia were added to the list in 1871.[†] He and Charcot also noticed, but did not publish, that when the right hemisphere was injured there was more emotional disturbance, and that of a depressed kind.

Mr. Callender,[‡] in his remarkable papers on brain shocks, noticed the difference in the effect of lesions of either hemisphere, and expressed himself as follows in his second conclusion:

"2. The rapidly fatal results of bleeding into the right hemisphere outside the thalamus and corpus striatum, as compared with bleeding into the corresponding parts on the left side."

Recently, De Fleury[§] has pursued the same inquiry with similar and more striking results. He adds to the above table, that sensibility is more often and more deeply impaired when the right hemisphere is diseased.

To sum up: Lesions of the right hemisphere give us

More frequent and greater anæsthesia.
Greater palsy.

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[*] C. R. de la Soc. de Biologie, 1870, pp. 27, 96, 116.

[†] C. R. de la Soc. de Biologie, 1871, p. 96.

[‡] Anatomy of Brain Shocks, in St. Bartholomew's Hospital Reports, iii., p. 415; v., p. 3.

[§] Du dynamisme comparé des hémisphères cérébraux. Paris, 1873.

Greater alterations of nutrition.	Optic neuritis (Jackson). Eschars. Œdema. Pulmonary congestion. Fever.

Palsy of sphincters.
Hysterical symptoms (emotional).

Lesions of the left hemisphere give us:

Less palsy and anæsthesia.
Aphasia.
Palsy of organs of articulation.

The general conclusions of this essay are:

First: I have brought forward facts to show that many hysterical symptoms may occur in diseases of the spinal cord and brain.

Second: That in diseases of the spinal cord these symptoms appear merely as a matter of coincidence.

Third: That in cases of cerebral disease the hysterical symptoms have a deeper significance, being in relation to the hemisphere injured.

NOTE.—*Nov.* 1*st*, 1874.—During the past summer and fall there have appeared in the *Lancet* a series of excellent clinical lectures by H. Charlton Bastian, on the common forms of paralysis from brain disease. In Lecture V., part 2 (*Lancet* of Sept. 26, 1874, pp. 440, 441), the author refers at length to the subject of difference of symptoms when either cerebral hemisphere is injured.

SYPHILITIC AND SIMPLE PACHYMENINGITIS.*

I PRESENT to-night a section of the diseased dura mater presented at the previous meeting by Dr. A. H. Smith, in order to prove not only that the diagnosis of gummy tumor was correct, but to exhibit some of the microscopic characters of the growth. I present also a second specimen, likewise a section of the dura mater, the lesions of which were simply inflammatory in character. It was removed from a gentleman of Texas who had been sent to me by Dr. Hadden, on account of persistent headache. Five weeks before that time the patient had been exposed to a very hot sun, and although not suffering from any of the symptoms of sun-stroke at the time, he subsequently was attacked with headache, from which he never entirely recovered. The headache was first seated in front, about the median line, over the region of the anterior fontanelle. It gradually extended backward in a symmetrical fashion, until it ultimately became seated in the occiput and back of the neck. His pulse was 55, and regular. He was examined very carefully, and nothing was found upon which organic disease of the brain could be based, and for the want of a better name the malady was called congestive headache. The patient went to the country, and, returning again in the fall, died.

I did not see him at the time, but had been informed that he had died comatose. There had been no difficulty of deglutition at any time, nor paralysis of any kind.

Dr. Clymer, who had seen the case in consultation, was reported as non-committal in regard to a diagnosis.

The autopsy was made by Dr. Hadden, and showed a large clot over the convexities of both hemispheres on the external surface of the dura mater, and enveloped between it and another membrane of new formation. This latter was formed originally as the result of inflammation, was embryonic in character, and contained a large number of newly forming blood-channels. This condition of things was quite satisfactorily shown under the microscope to the members. The opinion was expressed that one of these blood-channels had ruptured, producing the effusion in the substance of the new growth, giving the said effusion the gross appearances of a clot between two membranes.

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* A specimen presented to the New York Pathological Society, Feb. 9th, 1876. Reprinted from the N. Y. *Medical Record*, March 4; 1876.

A CLINICAL CONTRIBUTION TO THE STUDY OF POST-PARALYTIC CHOREA.[*]

HAVING had the opportunity of observing two well-marked instances of this remarkable symptom-group, and as I am able to bring forward one of the patients, I make bold to call the attention of the Association to the subject. Before reciting the cases, it may not be amiss to say a few words on the growth of our knowledge of the subject. Although the symptom-group had been observed a number of years ago by various physicians, yet it was our distinguished Fellow, Dr. Weir Mitchell, who first called special attention to it, and gave it a name, in 1874;[†] but already in 1873,[‡] Professor Charcot, of Paris, had quite well described the movements, and indicated their association with hemi-anæsthesia. I would here beg to call attention to the fact that the description of the symptoms in my first case was written in December, 1873. Recently M. Charcot has written specially upon "post-hemiplegic" chorea in its relations to hemi-anæsthesia,[§] and has expressed the opinion that both these symptoms are caused by lesions situated at the outer and posterior borders of the optic thalamus, or in the posterior expansion of the corona radiata. Last year,[‖] Dr. W. R. Gowers read to the Royal Medical and Chirurgical Society an elaborate essay based upon a number of cases showing various degrees of post-paralytic inco-ordination, including athetosis, athetoid movements, and post-hemiplegic chorea.

A good description of a remarkable case of this condition, also coincident with hemi-anæsthesia, will be found in M. Schœpfer's Paris thesis.[¶]

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[*] Reprinted from the Transactions of the American Neurological Association, vol. ii., 1877.

[†] Post-Paralytic Chorea. American Journal of the Med. Sci., Oct., 1874, p. 342.

[‡] Leçons sur les maladies du système nerveux, t. i, p. 279.

[§] Leçons, etc., 4me fascicule, Paris, 1877, p. 329.

[‖] On Athetosis and Post-hemiplegic Disorders of Movements. The *Lancet*, 1876, vol i., p. 709.

[¶] Considérations sur un cas d'hémianesthésie avec mouvements ataxiques, succédant à une hémiplégie du même côté. Thèse de Paris, 1876.

I now pass on to relate the cases which have come under my observation.

CASE I.—H. H. H., aged eighteen years; by occupation a clerk, and a native of this country, was referred to me by Dr. Fisher, December 6, 1873.

He was a fairly developed boy, with apparent good general health. He had been well until last April, when he began to suffer from diffused headache, more temporal, but not one-sided. One day in May he went down to his office feeling as well as usual, and began his work with full use of all his limbs. About 11 o'clock A.M. was writing, leaning on the desk, the left arm thrown forward and its fingers steadying the paper. He got up to cross the room with a book in his left hand, but the book fell to the floor, and he then first became aware that his left arm was weak. There were at the time no subjective or sensory symptoms, cerebral or peripheral. The leg was not in any way affected. He was able, though awkwardly, to feed himself at dinner that evening.

The paresis gradually increased until the middle of July, and since that time there has not been much change.

Shortly after the attack, within a few days, patient noticed numbness of the left hand and forearm, and this has somewhat increased.

In the early part of July convulsive movements began in the left hand, and have since become greater in force, and have extended to other parts of the left side. The left leg began to twitch and grow weak also in the first part of July, and since October slight twitching has appeared in the lower part of the left side of the face. Lately sight has become impaired, but hearing is preserved.

There have never been any symptoms on the right side. He has never had any epileptic or epileptiform seizures. Memory has somewhat failed.

In the middle of July there appeared double vision, which gradually passed away. At the same time he had severe bi-temporal headache with nausea, lasting one week. The latter symptom never recurred, and it was probably caused by diplopia. No rectal or vesical symptoms. No dysphagia.

EXAMINATION reveals left hemiplegia with peculiar spasmodic movements of the palsied parts. The left arm and leg execute all movements, though feebly and awkwardly. The dynamometer shows a strength of 30° in the right hand and 10° in the left; can barely stand on left foot alone; no evident facial paralysis; tongue points a little to the left. There is now no twitching in the face. The left pupil is a trifle larger than the right; both are active. The ophthalmoscope shows both optic disks congested, their outlines blurred, and traces of exudation along the blood-vessels. There is no evident palsy of any muscle about the eyes, though the eyes converge abnormally. Fields of vision not impaired.

There are strong *choreiform* (this is the word used in notes made at the time of first seeing patient—1873) movements in the left arm, shoulder, and leg. These parts are constantly agitated during waking hours, but quiet during sleep. The arm is more or less rigid, and its various parts perform large oscillations. The movement is increased by emotion or by the attempt to perform voluntary acts; he cannot carry a glass to his lips. Eyes being closed, he can, after groping a little, place fore-finger on tip of nose. There is, however, a marked ataxiform element in the spasm. The muscles of the shoulder and the trapezius are involved, but not the muscles of the trunk and neck.

Sensibility (to contact and pain) is slightly impaired in fingers and hands; the points of the æsthesiometer must be separated from 3 to 5 mm. to be distinguished on the finger-tips.

January 20th, 1874.—Chorea is as before. There is much hebetude. On the right side there is marked ptosis with weakness of the internal rectus (palsy of third nerve).

We now have a form of *crossed* paralysis, *i.e.*, third nerve on the right side, body on the left. The right crus cerebri is probably involved in lesion.

January 28th.—Greater evidence of palsy of third nerve on right side; more chorea in left limbs; left side of face paralyzed; speech thick, saliva escaping from the mouth. There is marked anæsthesia in range of left supra-orbital nerve. Sister states that he has frequent partial syncopal attacks without spasm or loss of consciousness. Is this petit-mal?

March 1st.—Much as before, but weaker. While speaking has spasm in left side of face which simulates involuntary laughter.

This patient died during March without presenting any new symptoms, and I was unable to make an autopsy.

Judging by the symptoms, the lesion must have been placed just above the motor tract of the right crus cerebri, acting upon it by pressure.

It is probable that the lesion was hemorrhage with subsequent inflammation about the clot, though it may have been a tumor. I am in doubt on this point, because I am not disposed to attach much value to the ophthalmoscopic examination which I made. The certain diagnosis of neuro-retinitis would, of course, have strengthened the probability of there being a tumor in the locality indicated.

CASE II.—J. P., aged 26, a clerk, was referred to me by Prof. Edward Curtis, on May 24th, 1877.

I learned that this young man had been well up to April 16, 1876, when he had, in the street, an attack of right hemiplegia. He did not fall down; was able to walk with help to a street car, yet knew nothing of what afterward occurred until the next day. This was either late loss of consciousness, or amnesia.

On the next day speech was much affected, he could not recall names and addresses of persons; his right arm was powerless, and there was much numbness of the right cheek and arm. "Could only see with inner half of right eye." Right leg weak. In a day or two speech improved.

In three weeks he walked well, but the arm did not regain its strength for three months.

During this period of improvement irregular movements appeared in the right arm, and have persisted. No jerking in face, leg, or on the left side of the body. Imperfect vision persisted. The numbness continued marked in toes, finger-tips, cheek, and tongue, on the right side. The numbness on the right side of face and tongue never quite reached the median line.

In August (four months after attack of paralysis) had an epileptiform spasm, followed by loss of consciousness lasting until the ensuing morning.

In April, 1877, had a second epileptiform attack. At times feels queerly in his head, "as if a spasm was coming on." Has not suffered from headache or dizziness.

Inquiries into patient's past history show that six or seven years ago he had several chancres, which did not heal for three months. Had no buboes. Never had any eruptions, strictly speaking; but his legs "ulcerated," and he has since frequently suffered from ulcerated sore throat. At no time any osteocopic pains. Sight was good until after attack of paralysis. Memory is a little impaired.

EXAMINATION.—Patient's speech is imperfect, a sort of aphasic stammering. It would appear that he has always had a somewhat similar defect. The right leg has almost perfectly recovered, but the right hand is not quite as strong as the left, and presents curious abnormalities of movements. The dynamometer test shows for the right hand 18°–22° ; for the left 28° and 28°.

The peculiar movements are of two sorts :

1. While the patient is seated quietly, with his hand resting on his thigh, it (the hand) may be seen to be agitated by slight rhythmical movements of the type observed in paralysis agitans. These are not truly constant, but seem to be provoked by observation or by the patient's own watching of the hand.

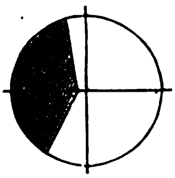
2. During attempted use of the extremity, an ataxiform movement is developed ; or, more properly speaking, a movement compounded of the want of combined and harmonious action of large muscular groups which is characteristic of the ataxic movement, and of the totally irregular and capricious muscular contractions which constitute the choreic type. On the whole the movement is more like that of ataxia ; *i.e.*, more or less regularly oscillatory.

The right leg is not the seat of any tremor, ataxia, or chorea ; and the left side of the body shows none.

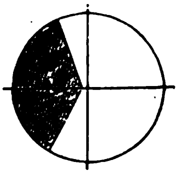
There is no marked facial palsy, but the right naso-labial crease is less distinct than the left. The tongue deviates a little to the right. These points make it probable that the paralytic attack in April, 1876, was one of common typical hemiplegia.

There exists a degree of right hemi-anæsthesia. To slightest contact, according to patient's positive statement, the anæsthetic district of the face begins at a point 1 cent. to the right of the median line. The æsthesiometer and the simple contact test show slight loss of sensibility on the right side of the face. On the finger-tips the points of the æsthesiometer cannot be distinguished at a less distance apart than from 5 to 8 mm. on the right side, while on the left they are distinguished at 3 mm. Pricking is well felt.

The fault in vision is in the shape of incomplete hemiopia ; the right temporal and the left nasal halves of the fields being obscured, as shown in the cut.



RIGHT EYE.



LEFT EYE.

The pupils are equal and normal ; the ocular muscles act well ; and the ophthalmoscope shows no lesion which I can appreciate.

A careful examination of the throat does not reveal any cicatrices. The heart is normal.

My diagnosis was slight cerebral hemorrhage just outside the left thalamus opticus. Yet I was unwilling not to give the patient the benefit of the doubt that syphilis had led to arterial disease and rupture, though the history was not one that pointed to the existence of syphilis at any time. Yet how often are serious syphilitic nervous lesions developed after equally or more incomplete chains of evidence? Consequently I have given this patient mercury internally, and have brought about a degree of salivation.*

REMARKS.—These cases are strongly in support of M. Charcot's proposition. In the first place, we observe in both the co-existence of hemiplegia, hemi-anæsthesia, and choreiform movements. In case I., a positive symptom, palsy of oculo-motorius nerve, points to a lesion near the crus cerebri—probably just above it. In the second, there is a strong probability that the lesion is not far from the same part—near the thalamus opticus.

In my second case, the occurrence of hemiopia is of great interest, because it will be remembered that Prof. Charcot has recently † denied that a lesion of the hemisphere could produce hemiopia. In the absence of a post-mortem examination, I would not be understood as claiming that the case positively contradicts M. Charcot's statement ; yet it must be admitted, I think, that there is no probability that there was in this case a second lesion affecting one of the optic tracts.

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* *July* 1, 1877.—I may now add that salivation, followed by the administration of iodide of potassium in doses of 12. a day, made no change whatever in this patient's symptoms. I have now advised him to cease treatment, and hope for spontaneous improvement.

† Leçons sur les localisations dans les maladies du cerveau. Paris, 1876, p. 126.

CONTRIBUTION TO THE STUDY OF LOCALIZED CEREBRAL LESIONS.*

It has fallen to my lot to observe during life and to examine after death a number of cases in which localized cerebral lesions gave rise to definite peripheral symptoms, and it has appeared to me that these cases might profitably be studied in the light of recent experimental and pathological researches upon the functions of the brain. In other words, I shall endeavor to determine the bearing of these cases upon the recent hypothesis of the localization of functions in the cortex of the brain.

I shall divide my cases into three categories: 1st. Cases in which localized lesions gave rise to Aphasia. 2d. Cases in which localized lesions gave rise to Paralysis. 3d. Cases in which localized lesions gave rise to Spasm.

PART I.

CASES IN WHICH A MORE OR LESS LIMITED CEREBRAL LESION PRODUCED APHASIA.

Case I.—*Cerebral softening from arterial degeneration; aphasia and right hemiplegia.*

A woman, aged seventy-five years, was admitted to the Epileptic and Paralytic Hospital on Blackwell's Island, January 17th, 1873. The history of the case is very meagre, and only states that when attacked she screamed, threw up her hands, and became insensible. On recovering from insensibility it was found that she had completely lost speech, and was paralyzed in the right side, face, and limbs. No mention is made of the state of sensibility; but, as I am in the habit of always looking for hemi-anæsthesia, I feel sure that the case was one of common hemiplegia. I also feel very positive that during the six months of the patient's stay in my ward she greatly regained voluntary power on the right side, being able to take a few steps alone, and to move her arm quite freely. Aphasia, however, remained complete.

The patient died on July 12th, 1873, and careful notes were made at the time of the post-mortem examination on the 13th. I reproduce only those relating to the state of the brain, making, however, the preliminary statement that the valves of the heart were free from disease.

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* Reprinted from the Transactions of the American Neurological Association, vol. ii., 1877.

Dura mater not abnormally adherent to the skull, and healthy at base. Moderate sub-arachnoid effusion at the top of the brain, and a great deal at its base.

Both anterior lobes have undergone more or less atrophy. The convolutions of the left parietal lobe, beginning at a point an inch and a half from the median line, bulge and form a soft and yellowish tumor. The pale yellowish color of the affected convolutions is in marked contrast with the injected appearance of the rest of the brain. Over these diseased convolutions there is more sub-arachnoid effusion than elsewhere. After removal of the brain, and escape of the greater part of the serum, the left parietal lobe appears sunken, and there results an appearance like a loss of substance as large as a small walnut. The basilar and other large arteries of the circle of Willis are open, and nearly free from changes.

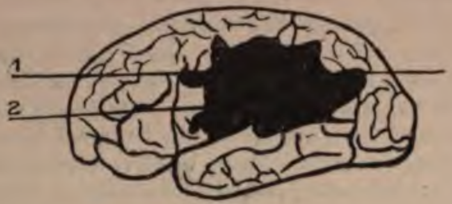


FIG. 1.

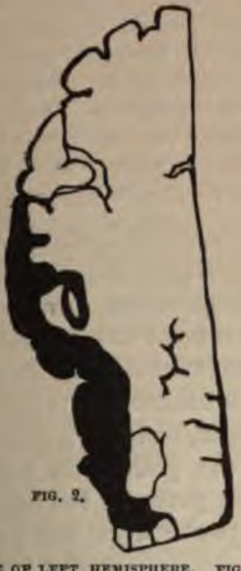


FIG. 2.

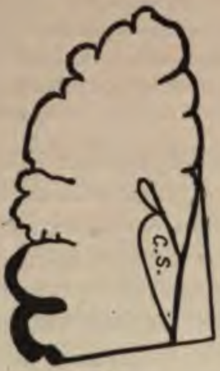


FIG. 3.

FIG. 1.—PROFILE OF LEFT HEMISPHERE. FIGS. 2 AND 3 TRANSVERSE HORIZONTAL SECTIONS OF SAME SHOWING EXTENSIVE RAMOLLISSEMENT.

Careful inspection shows no externally visible lesion in the left frontal lobe, its third convolution, and the island of Reil. The depression in the parietal lobe measures six and a half cent. square, involving all of the lobe except the part lying next to the great longitudinal fissure.

Two small old lesions are found in the right hemisphere, one lying at the external margin of the extra-ventricular nucleus of the corpus striatum, just behind the island of Reil; the other in the posterior part of the same (lenticular) nucleus. Both are small cavities, the size of large beans.

The following is a transcript of notes which I made upon the state of the left hemisphere. I was very careful in studying the limits of the *ramollissement*, because at first sight the case seemed to be one opposed to Broca's hypothesis of aphasia. The extent of the lesion was studied, after hardening for a few days in a solution of bichromate of potassa, by means of transverse horizontal sections, and tracings made on transfer paper upon each section by Mr. George Wright. Each tracing was afterwards carefully reduced and drawn to a scale, with a sketch of the appearance of the unsliced hemisphere. This last drawing, Fig. 1, shows that the greatest destruction of tissue has taken place in the parietal lobe proper. Posteriorly and inferiorly a degree of degeneration of the convolutions may be traced almost to the confines of the occipital and sphenoidal lobes; while anteriorly, after having destroyed in great part the ascending frontal convolution, it extends over a portion of the second frontal gyrus.

Sections were made through the hemisphere in the planes indicated by lines in Fig. 1, and the morbid appearances seen on each section, were accurately traced on paper. Fig. 2 shows that the atrophy has destroyed a large part of the upper posterior part of the hemisphere. Fig. 3 demonstrates that the hinder part of the third frontal convolution is likewise involved.

By this method of examination the case is restored to the category of common cases of aphasia, viz., those supporting Broca's hypothesis.

REMARKS.—I consider this case as doubly instructive.

First, in a positive manner, as showing that a superficial degeneration of the cortex of the brain (involving Broca's centre for speech) may produce aphasia of the most complete kind.

Second, negatively; because, in spite of the destruction of gray matter in the regions which Ferrier makes out to include nearly all the motor centres for the face, arm, and leg, a great degree of voluntary power was regained by the patient in a few months. Hence it would appear that other parts of the injured hemisphere had acquired controlling power over the limbs of the opposite side.

CASE II.—*Embolism of the left middle cerebral artery; softening of the brain; aphasia, and right hemiplegia.*

Mrs. G., seen in consultation with Dr. William Pierson, jr., of Orange, N. J., on May 8th, 1877. With the exception of a badly acting heart and a tendency to gout, Mrs. G. enjoyed fair health and had three children up to

1875. Late in November of that year she had a moderately severe miscarriage. On December 7th, at 7.30 P.M., experienced a sudden attack of common hemiplegia on the left side, without loss of consciousness. Speech was much impaired by defect in articulation. The palsy passed away rapidly, though she dragged her left foot for some days. Power of articulation slowly regained. Patient's husband is an unusually well-informed gentleman, and states that trouble in speech was surely not aphasiform. There remained a want of proper action of muscles of throat and larynx ; patient not knowing how to pitch her voice afterward.

On January 13th, 1876, a second attack of left hemiplegia occurred. Consciousness was lost for a moment. Left leg but not left arm palsied. All symptoms, except a degree of excitement, passed off the next day, after a long sleep.

On April 1st, at 1 A.M., had a first epileptic fit while asleep; had full spasm, slight frothing at mouth, loss of consciousness, stertorous breathing, and a heavy terminal slumber. On April 29th, second fit ; falling like a shot to the floor. Has had a few attacks since; only two, however, since last August. One was two weeks ago.

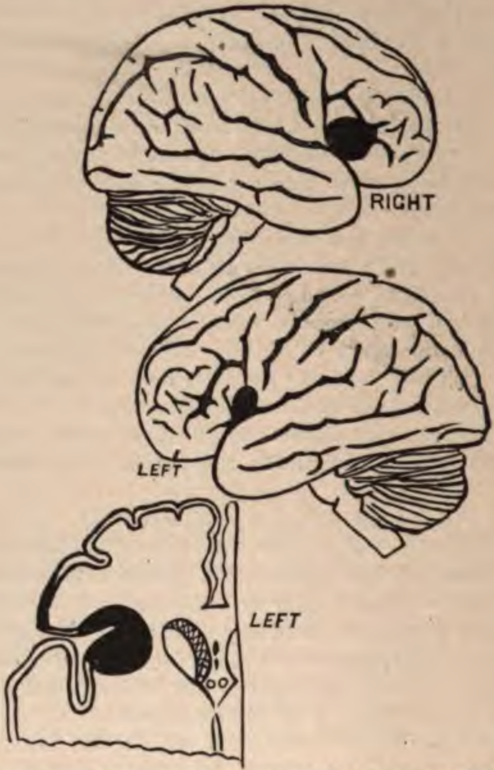
Present paralytic attack occurred twelve days ago. On April 26th, came down stairs, and in a few minutes was found fumbling a door-knob. Without apoplectic symptoms, complete right hemiplegia and aphasia were developed. Possibly a little return of voluntary movement has taken place in the right limbs. No return of speech. Indeed, the aphasia has been so absolute, that even sign language has been lost, and patient's friends have thought her deprived of her faculties, though she has been conscious at all times. There has been no retention of urine, no tendency to bed-sore. Food has hardly been taken, and patient's respirations have been extraordinarily frequent.

Examination shows complete right hemiplegia, with marked loss of sensibility in the paralyzed limbs. The face is hardly distorted. Absence of spoken and gesture language as above, but patient looks intelligent. Pupils very wide, but equal. Breathing very rapid, 50 or 60 in the minute. Pulse slightly irregular, beating about 90 in the minute. The heart is enlarged, apex in sixth intercostal; cardiac sound can be heard away from the patient, as far as two feet. No distinct murmur can be heard, but the heart sounds are completely reduplicated. Oral temperature 37.5° C. No sensibility to pinching on the right side, and only slight on left; but this may be owing to rapid respiration. There is a marked tendency to turn eyeballs to the left, away from the paralyzed side. Lungs are free from deposit or congestion; the optic disks are normal.

I learn that Mrs. G. has had pains in the small joints, with atrophy of muscles of the hand, and, in the last few years, gouty knees. From childhood she has had a curious affection of the heart, characterized by dyspnœa, hard beating of the organ, and by transmission of its sound to a distance from the body of two to four feet. My diagnosis was embolism of various arteries in both hemispheres; the last attack being due to blocking of a branch, or branches, of the left Sylvian artery, and consequent softening of (probably) the third frontal and the ascending frontal convolutions.

In spite of an attempt at supporting treatment, Mrs. G. died on May 11th. For notes of the post-mortem examination, made on the same day, six hours after death, I am indebted to Dr. Pierson.

"The membranes and sinuses of the brain were normal. The surface of the third frontal convolution of the right side was yellow in color, with a tough, elastic feel. This lesion, extending through the gray matter, was about 3. cent. square and .5 centimeter in thickness. The same change was also found, but to a less extent, in the same part on the left side. The left middle cerebral artery was plugged by an embolus at a point 1.25 centm. from its origin. A spot of cerebral tissue, about the size of a quarter of a dollar (3 centm. in diameter), supplied by this artery, was in a softened state; it was yellow, tinged with red, broken down, and of semi-fluid consistence. The situation of this lesion was external and posterior to the anterior cornu of the left lateral ventricle, and corresponded to that part of the third frontal convolution which is known as the island of Reil. The softening was confined to the white substance, and did not implicate the surface. The rest of the encephalon was normal.



CASE II.—SOFTENING IN BOTH HEMISPHERES, IN CONSEQUENCE OF EMBOLISM.

"The heart was much diseased. The mitral valves were much thickened, and nodules of atheroma could be felt at various points. The two leaves of the valve were joined together, constricting the auriculo-ventricular opening to such an extent that it would not admit the end of the finger; there was stenosis and insufficiency."

Unfortunately the brain was not preserved for more minute examination, so that the existence of microscopic changes in the rest of the territory supplied by the left Sylvian artery remains a matter for speculation.

The notes and the plugged artery were brought to me by a student of Dr. Pierson, and I asked him to mark out for me on plates in Ecker's and Luy's books the location of lesions. The accompanying diagrams are constructed faithfully after this gentleman's indications, and Dr. Pierson's corrections.

REMARKS.—The complete aphasia in this case was undoubtedly due to lesion of the third convolution and of the anterior folds of the island of Reil. We have in this case an addition to the large list of cases supporting Broca's hypothesis. It is remarkable that such complete hemiplegia should have existed without apparent lesion of those parts which are more directly connected with the movements of the arm and leg, viz., the ascending frontal and parietal convolutions. These convolutions are supplied by the artery which was plugged, and it is very probable that microscopic study would have shown them to be full of granular bodies. They may have been saved from gross softening by an unusually free anastomosis between the final branches of the plugged Sylvian artery and other arteries in the same hemisphere. It is also possible that a part of the hemiplegia was due to pressure upon and anæmia of parts adjacent to the softened spot; and that, had the patient survived, the paralysis would have disappeared in a few weeks.

Another interesting feature of the case lies in the connection between the very localized lesion in the right hemisphere and the first attack of hemiplegia. This attack, it will be remembered, was characterized by slight and transitory paralysis of the limbs, and by very great defect in articulation. Indeed, it is said that the patient never recovered the full use of her vocal organs. From this we might be led to infer that the function of the third frontal convolution on the right side in right-handed human beings is intimately connected with the muscles governing the movements of articulation and phonation.

CASE III.—*Chronic meningitis almost limited to the posterior part of the third left frontal convolution; extensive central cerebral soften-*

ing; epilepsy, chronic aphasia of varying degree, right hemiplegia; death in status epilepticus.

A male, age forty-seven years, was admitted to the Connecticut General Hospital for the Insane, at Middletown, on November 29th, 1873.

For a full account of the case and for the post-mortem examination I am indebted to my friends Drs. A. M. Shew and W. B. Hallock, medical officers of the hospital.

The patient was sent to the hospital because of "dementia." It was learned from his wife that he had had three "apoplectic" attacks. The first occurred in April, 1873, and consisted in loss of consciousness and general lspasm. After this he had at short intervals some few slight "spasms." In July had a second severe attack of same kind as the first, followed by slight hemiplegia. Three weeks later had a third seizure, after which he was in a state of delirious mania, which still continues.

On admission, mind is in dementia, the pupils are very small, articulation good, the tongue protrudes straight, and the only palsy apparent is in right lower limb. At times is violent and has insomnia. Speech very incoherent. In December it is noted that palsy is less, but that legs are not well co-ordinated, and that hands tremble.

In middle of January, 1874, patient is calmer, and more rational, but speech is imperfect. "He talks plainer, seems to have ideas, but has forgotten words." Aphasia.

Feb. 17th.—At two o'clock P.M. had an epileptic attack, with paralysis of right side; tongue deviating to the right. Four other spasms in three-quarters of an hour; each characterized by usual symptoms, foaming at mouth, stertor, upturned eyeballs.

March 6th.—Is up and about the ward. Is more coherent than for some time. Hallucinations of sight in the night.

March 30th.—Is discharged improved. For some time past the difficulty in speech has taken the form of amnesia, and his writing has shown the same characteristics.

Re-admitted June 29th, 1874. Aphasia still present, and he seems to appreciate the trouble. "I like that fountain I drink from three times a week," meaning I like that medicine I take three times a day. He knows that he calls things by wrong names and is thereby irritated. "I wish you would sell me," meaning bleed me. "Oh, I can't talk."

July 26th.—Yesterday and last night had ten attacks of an apoplectiform character, without paralysis. These must have been epileptic seizures.

Sept. 29th.—Rose as usual, made up his bed nicely. At table was unable to grasp knife and fork. In twenty minutes complained of bad smell and dim vision, then had a general convulsion. Had ten attacks in the course of an hour. There was foam at the mouth, and more twitching on the right side of the body. In a few days was up again.

Oct. 20th.—Three epileptic attacks in a few minutes. This is the third time that patient has had prodromata, consisting of nervousness, a slight degree of paralysis of right side; has gone to his room and made signs that he was ill.

Oct. 21st.—No paralysis after attacks; some excitement in night.

Nov. 19th.—Is in good physical health; has hallucinations of sight; aphasia continues marked.

Jan. 8th, 1875.—After dinner (1 P.M.) had an epileptic seizure, and at 5 P.M. the attacks followed each other rapidly. The right side is completely paralyzed.

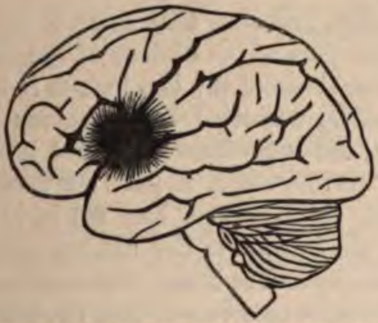
Jan. 9th.—Has been in status epilepticus all night, and must have had nearly four hundred convulsions. Died at 3.15 A.M.

Autopsy Jan. 10th, thirty-two hours post mortem. Calvarium normal. Vessels of dura mater and of pia mater congested. Dura mater adherent to bone over right posterior lobe of cerebrum, membranes adherent to pia mater over left middle lobe of brain. General moderate opalescence of the arachnoid. Other organs in remarkably good condition.

The entire brain was at once sent to me in a solution of bichromate of potassa, and I made a careful examination of it in a few days.

The only externally visible lesion is on the left hemisphere. There a patch of dura mater adheres to the soft membranes and the brain, at the posterior extremity of the third frontal convolution and the lower extremities of the ascending frontal and ascending parietal convolutions, bridging over the fissure of Sylvius. The patch measures 22 mm. in a vertical direction, and 15 mm. horizontally. The anterior border of the piece of membrane is 58 mm. from the apex of the frontal lobe; its upper edge is 58 mm. below the upper border of the frontal lobe (at the longitudinal fissure); and its lower border, lying on the fissure of Sylvius, is 55 mm. above the lowest part of the sphenoidal lobe.

The arachnoid and pia mater surrounding the patch, in the fissure of Sylvius and for 25 mm. above it, are thickened and whitish.



CASE III.—PATCH OF PACHYMENINGITIS ADHERENT TO LEFT HEMISPHERE.

The patch lies in a depression in the underlying convolutions, and a horizontal transverse section through it and the hemisphere shows that the three membranes are fused in the patch, which is 2.5 mm. thick, and that the subjacent gray matter of the third and ascending frontal convolutions, and the ascending parietal convolutions, are grayish and translucent.

The same section reveals extensive softening in the central parts of the left hemisphere. The lenticular ganglion (extra-ventricular part of corpus striatum) is soft and reddish; contains nervous débris, granular bodies, and granular blood-vessels. The few ganglion cells seen are filled with granulations.

A second softened spot is in the white centre of the hemisphere, in its posterior half, outside and above the lateral ventricles. The microscope shows the same granular detritus as in lenticular ganglion. Abundant granular bodies are also found in the white centre of the frontal lobe, and in the convolutions of the island of Reil, deep in the fissure of Sylvius.

The corpus striatum proper contains only a few granular ganglion cells.

Microscopic examination gave evidence of descending degeneration in the motor tract through the left half of the pons Varolii and medulla oblongata, and the right lateral column of the spinal cord.

The ganglion cells of the anterior horns of the spinal cord contained an abnormal amount of granular matter, and the posterior columns contained an immense number of amyloid bodies.

REMARKS.—This case, partly from its complicated nature, and partly because of its imperfect record, is difficult to analyze with reference to the question which I have in view in this contribution.

In addition to epileptic, paralytic, and aphasic manifestations, there seems to have been actual insanity present, as manifested during life by hallucinations and delusions, and by incoherence and dementia; and post-mortem, by opacity of the delicate membranes of the brain, and degeneration of the posterior columns of the spinal cord.

It seems probable, however, that some of the so-called "dementia" in the first part of the history of the case was aphasia.

In estimating the share of the two lesions in the production of symptoms it must be admitted that, whereas the patch must have been ancient, the *ramollissement* cannot have been very old. The latter lesion was, however, at least six weeks old, since secondary descending degeneration had set in to a slight degree. Yet, as the chief phenomena of the disease—epileptic seizures, transitory hemiplegia, and aphasia—existed from the beginning of the illness, it is right to conclude that the older lesion, *i.e.*, the localized chronic meningitis, was the cause of these symptoms.

The thickened state of the pia mater, arachnoid, and dura mater acted upon subjacent parts in several ways: by pressure and by mechanical irritation, increased by the respiratory and cardiac movements of the brain, and by interfering with the blood-supply of the gray matter, and of the white substance for a certain depth. The part which was most affected by this lesion was

Broca's speech centre in the posterior part of the third frontal convolution.

Consequently, it seems right to me to consider this case as favorable to the hypothesis of the localization of functions in limited parts of the cortex of the brain.

As to the nature of the localized meningitis, nothing is said in the history of the case concerning syphilis, or symptoms belonging to the syphilitic category. Sections of the patch and subjacent nervous tissue were very difficult to make, because of the difference of density between the two tissues and the lax bond of union between them. The specimens now passed around must be judged leniently because of these peculiarities. In so far as the thickness of the sections will permit microscopic study, it seems to me that the lesion is a simple hyperplastic one, having resulted in the formation of dense fibrillar connective tissue. In no part of the specimens can I find the numerous young cells so characteristic of gummatous products.

CASE IV.—*Constitutional syphilis, commencing caries of the dorsal vertebræ, acute tuberculosis; tubercular meningitis, most developed over left third frontal convolution and island of Reil; intermittent aphasia, and later, hemiplegia.*

Mr. X——, aged forty-three years; a private patient of Drs. William H. Draper and Frank P. Kinnicutt. The latter gentleman has kindly furnished me with abbreviated notes of the case, and the brain was placed in my hands for examination.

The patient was a victim of unusually severe syphilitic infection: having had series of secondary and tetiary lesions while under Dr. Draper's care. At the time of his last illness he had syphilitic neuralgia. In 1874–5 there was slight trouble at apex of right lung, but the disease seemed wholly arrested during the past year.

Since two months, emaciation has rapidly advanced, and strength has much diminished. Since April 20th there has been fever of a very irregular type; the temperature varying from 37.1° to 39.1° C.; the temperature not being the same on any two days. Pulse has been very frequent: 100 to 120. Physical examination has revealed simply moderately fine moist râles, at first only in the anterior and posterior parts of the right lung; later, during the last four weeks of life, in both lungs.

On May 10th there was suddenly developed aphasia and agraphia, without loss of consciousness or paralysis. [This negative statement is not made upon the patient's or the nurse's statement, but after critical examination by Dr. Kinnicutt, whose accuracy in clinical observation is extreme.] This condition of aphasia continued about twenty-four hours, and during this time the patient's mind was perfectly clear. A nearly complete intermission (return of speech) then occurred, followed in twenty-four hours more by a second attack of complete aphasia and agraphia, also without impairment of consciousness or mental clearness, and without paralysis. These intermissions and attacks continued to succeed one another until within forty-eight hours of death, when the intermissions became incomplete. At first there existed only a certain slowness of speech during the intermissions; but in the last few days there was partial aphasia; or, more properly speaking, aphasia would show itself after a few moments of correct speaking. Right hemiplegia was almost imperceptibly developed (face and limbs); became marked forty-eight hours before death, and was complete, with a semi-comatose state, at the last.

At no time was there only pain in the head. General hyperalgesia (more marked on the right half of the body) was present in last week of life.

Dr. Kinnicutt diagnosticated acute pulmonary tuberculosis, with a cerebral complication. There was a doubt on our minds whether the cerebral lesion was meningitis, tubercular or syphilitic, or whether it consisted in syphilitic arteritis, involving the left middle cerebral artery.

The post-mortem examination was made sixteen hours after death, by Dr. Kinnicutt, and the following lesions found:

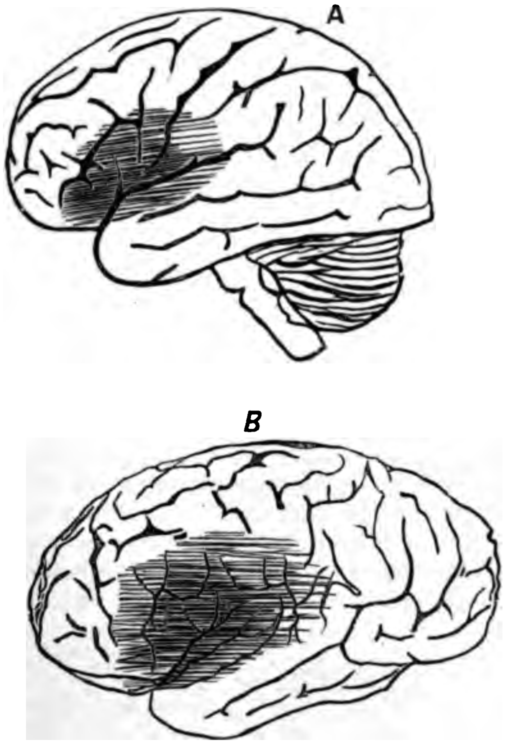
There was a cavity about two centimeters in diameter, filled with pus, in the right half of the body of the twelfth dorsal vertebra; and the pus was seen to have made its way within the sheath of the psoas muscle, nearly as far as Poupart's ligament. At the apex of the right lung was a dense cicatrix, and over one of the lower ribs on the left side the remains of a gumma which had suppurated many months. Gray tubercular granulations were found in abundance throughout both lungs, in the spleen and kidneys. There were none in the liver, and their presence in the peritoneum was doubtful.

The brain, placed in a solution of bichromate of potassa, was at once sent down to me for examination. I did not do more at first than note that the left sphenoidal lobe had been damaged in the removal of the brain, and that there were a very few granulations, the size of tobacco seeds, or a little larger, in the pia mater on the convexity of the hemispheres. My reason for not cutting the brain while fresh was that, from the lack of consistency of the left anterior lobe, Dr. Kinnicutt thought it likely that there was an abscess or a patch of *ramollissement* near the third frontal convolution, and I wished, if there were such a lesion, to obtain its exact topography.

The brain having been completely hardened in bichromate of potassa, I proceeded to examine it by means of vertical and horizontal sections. Somewhat to my surprise, I found no lesion in the deeper parts of the hemispheres. There was only a leptomeningitis of very peculiar distribution.

In the first place, the convexity and base, and the whole of the right hemisphere showed only traces of exudation alongside of the chief vessels of the pia mater, with here and there a granulation varying in size from .5 mm. to 1 mm. in diameter.

In the second place, the lower median region of the left hemisphere showed very much more developed exudation. The vessels covering the posterior part of the third convolution as it dips into the fissure of Sylvius were bordered by thick bands of exudation quite as wide as the vessel itself, and the pia mater was thickened over a space 25 mm. or more in diameter. On opening the fissure of Sylvius, the pia lying in it was found enormously thickened, and its meshes filled with semi-solid and solid exudation.



CASE IV.—A. SHOWING FOCUS OF TUBERCULAR MENINGITIS ON EXTERNAL PART OF LEFT HEMISPHERE. B. THE EXUDATION IN THE FISSURE OF SYLVIUS.

The focus of the meningitis was in the territory of distribution of the left middle cerebral artery, especially over the third frontal convolution, and the convolutions of the island of Reil.

Microscopic examination of the exudation showed an accumulation of young cells in the meshes of the pia mater, and more especially round about blood-vessels next the cortex. Around many of these the exudation formed tumor-like swellings, or, more exactly speaking, muff-like masses. The young cells could be followed some distance into the cortex of the brain, lying in the perivascular spaces. This examination corroborates the diagnosis of tubercular meningitis.

PART II.

CASES IN WHICH A LIMITED CEREBRAL LESION CAUSED PARALYSIS.

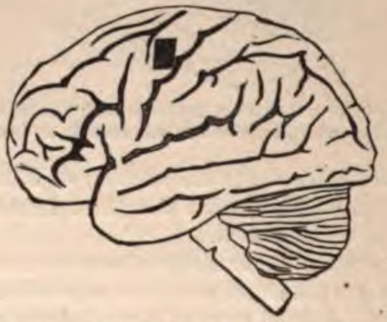
CASE V.—*Limited softening of the left ascending frontal convolution; right hemiplegia without aphasia.*

A female, aged fifty-four years, was admitted to my ward in the Epileptic and Paralytic Hospital on Blackwell's Island, on June 20th, 1875.

The brief history of her case is to the effect that about Christmas, 1874, during the night, she was suddenly paralyzed on the right side. She did not lose consciousness, her face was not paralyzed, and speech was preserved.

Examination shows that patient's face is not paralyzed, her tongue points straight, the right upper extremity is palsied, but there is slight voluntary motion at elbow and fingers. The lower limbs are without voluntary motion. There is some œdema about the ankles, and a superficial bed-sore on both nates. Death occurred June 23d.

Post-mortem examination.—The only externally visible lesion in the brain is a disappearance of a part of the convolution in front of the fissure of Rolando, within 25 mm. of the great longitudinal fissure, on the left hemisphere. It is not like a yellow patch, but is more like an ulceration of the convolution. A horizontal section, made through the hemispheres above the ventricles, shows healthy tissue in the right hemisphere, while in the left it reveals a softened but not much discolored part in connection with the superficial lesion above described. A vertical transverse section through this lesion and the lower part of the left hemisphere shows that the softened mass has its greatest diameter vertically, and extends from the roof of the lateral ventricle upward.



CASE V.—LIMITED SOFTENING OF THE LEFT ASCENDING FRONTAL CONVOLUTION.

No other lesion is found in the cerebral tissue. The cerebral arteries show some arteritis, especially the left middle cerebral; but their channels are open.

A considerable spinal lesion is found, consisting of purulent infiltration of the lower cervical muscles alongside the 3d, 4th and 5th vertebræ. On removing the posterior portion of the vertebræ, there is found a thick exudation, like a membrane, between the dura mater and the bones. Between the dura mater and the bodies of the vertebræ there is no trace of exudation. The arachnoid and the spinal cord seem healthy. The only osseous lesion is a roughness of the arches of the cervical vertebræ near the exudation.

This case is only to be made use of with reservation, because of its imperfect history, and because of the co-existence of a spinal peri-pachymeningitis. The main interest of the case is a negative one of unquestionable exactness. At no time was there aphasia; and it may be seen that the softened patch was placed quite far from the speech centre, above and behind it. It would seem that the face and tongue were not palsied; and this again is in accord with the location of the lesion, which is in the ascending frontal convolution, near Ferrier's centre, No. 6, which he[*] finds in the monkey to co-ordinate certain movements of the arm and forearm.

PART III.

CASES IN WHICH LOCALIZED CEREBRAL LESIONS GAVE RISE TO LOCALIZED CONVULSIONS OR SPASM.

CASE VI.—*Injury to the top of the skull on the right side, osteitis, inflammation of the dura mater, with lesion of subjacent convolutions; development of a large sarcomatous tumor in the right hemisphere; epilepsy; spasms limited to left arm, neck, and face; left hemiplegia; no neuro-retinitis.*

George S., a laborer, was admitted to my service in the Epileptic and Paralytic Hospital, Blackwell's Island, on April 19, 1875, for epilepsy.

The following history of his case was obtained:

On December 19, 1869, he got out of bed, in the middle of the night, to get some water; went to a stairway on the outside of the house, made a misstep, and fell to the ground. He lay insensible until morning, when he was taken care of. Paralysis of the left side followed the injury, but improved under treatment sufficiently to allow him to do his ordinary work.

Three years later (1872), he was seized with epileptic convulsions of the common typical sort: sudden fall, general spasm, biting of tongue. These attacks ceased in December, 1874, and were then wholly replaced by very frequent attacks of partial or localized epilepsy, without loss of consciousness.

These attacks consist of tonico-clonic spasm of the muscles of the left side of the face and neck, and of left upper extremity, especially the thumb and index. The left upper extremity is strongly flexed and the mouth drawn to the left during the attack, which lasts from 80 to 140 seconds without nitrite of amyl, and from 70 to 90 seconds with it. The attacks occur with extreme frequency, from three to eleven taking place every hour. At night they are rare. The spasm begins simultaneously in the facial muscles and in those governing the thumb and index. Consciousness is never lost. No general convulsions occur. An examination shows the left pupil to be a trifle larger than the right; the left cheek is paretic; the left arm and forearm absolutely paralyzed, and the left leg weak. The gait is characteristic of hemiplegia. There is marked tactile anæsthesia on the left side; the two points of an æsthesiometer not being distinguished at 45 mm. on the forehead, at 40 mm. under the eye, at 40–50 mm. on the side of the cheek and on the fingers. He feels pricking normally on the face and fingers.

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[*] The Functions of the Brain. New York, 1876, p. 306.

The injury to the skull, caused by the fall in 1869, is indicated by an irregular depression existing on the vertex, within an inch to the right of the median line, in a plane passing vertically through the external auditory meatus.

The attacks of limited epilepsy were much reduced in frequency, but not interrupted for any length of time by the systematic use of the bromides pushed to the limit of prudence. The partial hemiplegia increased.

June 28th.—The weakness of the left leg has greatly increased; patient is unable to stand or walk without help; the fingers can be moved a little, and a degree of contraction has appeared in the elbow and hand.

Sept. 13th.—Patient had a slight general convulsion, with loss of consciousness, last night.

December 3d.—Is growing gradually worse. The ophthalmoscope shows fullness of veins, but no neuro-retinitis. Patient lies on his back, with left arm strongly flexed; there is some opisthotonus; complains of being deaf in right ear. Axillary temperature 36.4° C.

December 7th.—Complains of pain in right arm and leg, and in posterior part of head on the right side; pupils are equal and of medium size. There is no distortion of face, and the tongue comes out straight. The left upper extremity is completely paralyzed; the forearm and hand flexed and rigid. Left lower extremity is deprived of voluntary power, and lies rigidly extended. The neck is rigid, with tendency to opisthotonus. During the examination (and at other times) patient turns his head and eyes away from the palsied side—*déviation conjuguée.* Contact and pinching are felt on the palsied side. Pinching produces reflex spasm in the left limbs, of the nature of spinal epilepsy, *i.e.*, tonico-clonic spasm. The veins of the neck and forehead, especially on the right side, are unnaturally distended. An ophthalmoscopic examination shows no neuro-retinitis. Pulse 108, axillary temperature 37.1° C.

Localized and general convulsions recur from time to time. Patient is semi-conscious.

Death occurred on December 23, 1875.

The body is much emaciated, and rigor mortis is well marked, especially on the left side. On removing the scalp there occurs a large escape of blood from enormously distended veins. The occipital veins on the left side are just perceptible, while on the right side they are 6 mm. in diameter. The frontal veins on both sides are much developed, but not filled with blood, because the body has lain upon its back. The right frontal bone is the seat of marked irregularities, and is rough and fissured between the median line and the external angular process. On the top of the skull, on a line passing vertically in front of the external auditory meatus, and 12.5 mm. from the median line, are two depressions about the size of peas. One of these contains a varicose vein, and a sound passed through the aperture seems to strike against the dura mater.

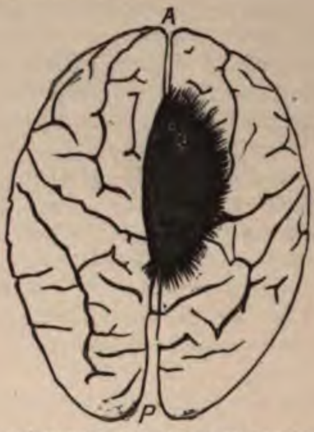
After sawing the skull through in the usual manner, the brain is divided horizontally without removing the skull-cap. The inferior half of the brain, examined as it lies in the fossæ of the skull, presents the following peculiarities: The right half of the brain is much enlarged, and the lateral ventricle and septum lucidum are forced over to the left. On the left side, the corpus striatum, thalamus, and convolutions appear healthy. On the right side the intra-ventricular nucleus of the corpus striatum alone is normal. Its extra-ventricular nucleus is undistinguishable. The white matter forward and outside of the corpus striatum, and in the neighborhood of the optic thalamus, is of a creamy consistency. The external and posterior half of the thalamus is involved in this softening. The convolutions directly outside of the thalamus are very much crowded together. At the base of the brain the convolutions lying immediately over the right olfactory bulb are very much softened, as are also the convolutions of the right sphenoidal lobe. The inferior convolutions of the occipital lobe appear normal. The white matter of the apex of the right anterior lobe is so soft that it is torn through in removal.

The upper half of the cerebrum, still lying in the skull, appears as follows: The septum lucidum is displaced to the left of the median line fully one-half inch. The white substance of the right hemisphere is the seat of a tumor, larger than a hen's egg, movable in the substance of the hemisphere, and of the consistence of glandular tissue. The opto-striate bodies of the right side are strongly compressed by the tumor, and so are the convolutions of the parietal region. The white matter in front and behind the growth is softened.

The upper half of the brain is carefully detached from the calvarium, by pushing the fingers between the dura mater and the bone. No difficulty is experienced in doing this until the neighborhood of the external depression in the bone is reached, when the dura mater is felt to be strongly adherent to the skull. With some difficulty the adhesion is broken up and the brain removed.

A careful examination of the convexity of the brain, thus exposed, shows the dura mater depressed and firmly adherent to the convolutions on the right side of the longitudinal fissure. There is some bony formation in the depressed adherent part of the dura. Around the patch, which is 25 mm. in diameter, the dura mater is moderately adherent to the anterior lobe over an oval space, measuring 75 mm. longitudinally, and 38 mm. transversely from the median line. The longitudinal fissure opposite this patch is obliterated by adhesions, and these are also present 13 mm. to the left of the median line.

Just to the right of the median line, opposite the depressed adherent spot in the dura, the skull presents a marked thickening and roughness. This tumor-like development of bone is 25 mm. anterior to the opening in the skull above described. A transverse section of the bone at this point shows it to be 13 mm. thick, and much condensed.

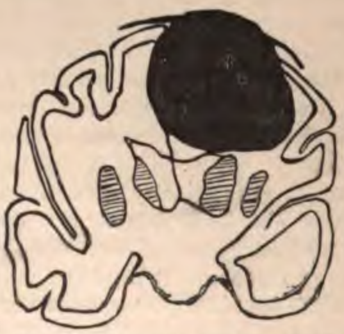


CASE VI.—PATCH OF THICKENED DURA MATER ON TOP OF RIGHT HEMISPHERE.

Transverse sections through the pons Varolii show no lesion except hyperæmia.

The fossæ of the skull are unusually deep, and irregularly furrowed and hilly.

The upper half of the left hemisphere, after hardening in bichromate of potassa, is examined by means of vertical transverse sections. The surfaces of these sections show that the tumor is very much larger than would appear from an inspection of the convexity of the hemisphere, or of the horizontal section above the ventricles. In fact, almost the whole of the anterior two-thirds of the upper half of the right brain is occupied by the growth, which has partly pushed aside and partly taken the place of the nervous tissue.



CASE VI.—TRANSVERSE VERTICAL SECTION THROUGH HEMISPHERES, SHOWING RELATIVE SIZE AND POSITION OF THE TUMOR.

A transverse vertical section made through the anterior third of the thickened patch of dura mater reveals that the tumor occupies the whole thickness of the hemisphere above the opto-striate bodies, and that these bodies are strongly pressed upon. The mass of the tumor is so considerable as to press upon the inner surface of the left hemisphere.

A transverse vertical section through the middle of the thickened patch of dura mater and the cerebrum shows an appearance represented in Fig. 2. The tumor, continuous with the dura mater, extends deeply into the substance of the right hemisphere, down to the level of the roof of the lateral ventricle. Besides, this roof and the falx cerebri are much displaced by the growth.

Posterior to this level the tumor diminishes rapidly in size, though still pushing over to the left of the median line.

A transverse vertical section made in the hinder part of the brain (beyond diseased dura mater) shows the tumor only as a small nodule in the upper inner part of the section-surface, lying wholly in the white substance.

The great longitudinal sinus is obliterated for a space of nearly 50 mm. in the thickest part of the patch of dura mater.

Microscopical examination showed the tumor to be an alveolar sarcoma in parts, and in others a common sarcoma.

As regards the connection between these lesions and the symptoms during life:

These symptoms were at first epilepsy, for nearly two years, followed by partial hemiplegia on the left side of the body, by incomplete localized epileptiform spasms in the left cheek, neck, and upper extremity, and the scene closed with complete left hemiplegia and a few general epileptic attacks.

It seems right to me to connect the above symptoms with the lesions in the following manner:

The first manifestations, general epileptic seizures, were caused by the development of thickening of the skull (internal plate), inflammation of the dura mater, and irritation of the cerebral substance.

As the pachymeningitis increased, with formation of bony spicules in its substance, the inflammation extended along the pia mater farther outward and backward, and thus reached parts of the convolutions which lie next to the fissure of Rolando and above the upper end of the fissure of Sylvius. These parts cover the regions numbered 2, 3, 4, and 6, in Ferrier's chart of the probable motor centres in the human brain, as deduced from experiments on apes.

By this irritation, there were caused the peculiar spasmodic movements of the muscles on the left side of the face and neck and of the left hand and arm. Ferrier considers the regions numbered 2, 3, 4, and 6, as motor centres for the hand and arm chiefly.

There was also partial left hemiplegia. Later still, sarcoma was developed from the pachymeningitis; the malignant growth rapidly extended in all directions, substituting itself for the nervous tissue, and producing powerful compression-effects in all directions, but chiefly downward upon the opto-striate bodies.

In this terminal stage of the disease, the phenomena were complete left hemiplegia, a few general convulsions, no localized spasm.

As an additional sign of extensive lesion of the hemisphere, we may note the conjugate deviation of the eyes, away from the paralyzed side, and toward the injured hemisphere.

Why, with such an enormous tumor, and such an increase of intra-cranial pressure as must have existed, there was no amblyopia, neuro-retinitis, atrophy of the optic nerves, diplopia or hemiopia, is a very puzzling question. This case is the second one of large cerebral tumor without choked disks or atrophy of the optic nerves which I observed in 1875.

In conclusion, I think that this case may be looked upon as corroborative of Hughling Jackson's, and Ferrier's theory of the existence of excitable motor districts, in some way connected with the motions of the face and forearm, in the upper median convolutions of the cerebrum—the ascending frontal and ascending parietal convolutions.

CASE VII.—*Pneumo-pyo-thorax; suppurative cerebral meningitis; abscesses in both hemispheres; localized epileptiform spasms in left hand, arm, and face; no paralysis or aphasia.*

For notes of the following remarkable case I am indebted to my friend, Dr. F. P. Kinnicutt.

The patient, a lad of thirteen, had been under the care of Drs. William H. Draper and Kinnicutt for empyema during several years.

During the last year of his life, the patient had a number of attacks of what was called slight septicæmia, characterized by moderate chill, fever, and diminished secretion from the pectoral fistula. The checking of the outflow of pus was held to be the cause of these attacks.

On February 18th, 1877, there occurred a severe chill, followed by fever, with a temperature of 37.7° and 38.8° C., lasting forty-eight hours. There was severe diffused headache. Dr. Kinnicutt was called and made the following observations:

On February 23d, patient felt very well, and it was thought that this attack had terminated favorably, like the others. But suddenly, while engaged in play, there occurred a twitching in the middle finger of the left hand; the spasm soon extended to the forearm. The entire spasm (clonic in form) only lasted a few seconds, and rather amused the boy. A few hours later a second attack took place, and was witnessed by Dr. Kinnicutt. Patient was aware of beginning of spasm by an almost imperceptible tremulousness of the facial muscles on the left side, followed, a few seconds later, by clonic spasm of the left middle finger and thumb (the former being strongly flexed, the latter adducted and flexed), succeeded by clonic flexions of the forearm, and evident clonic spasm of the left side of the face. The entire seizure lasted about 60 seconds, and during it there was no spasm of any part but the left face, arm, forearm, and fingers. There was no loss of consciousness, and patient conversed intelligently during the attack. The pupils and vision remained normal. Not a trace of local or general paralysis followed the spasm (contrast to Hitzig's case). There was no anæsthesia or abnormal sensation (aura or numbness) in the affected parts before, during, or after the attack.

The headache, of which he has complained since the 18th, has grown much worse; it is not localized, but is more violent in the occipital region; the pain extending into the nape of the neck.

There were five or six of these hemiplegic spasms on the 23d.

I saw the patient in consultation with Dr. Kinnicutt that evening, and a careful examination failed to reveal any objective symptom except a buccal temperature of about 37.7° C. The pulse was proportionately rapid, but not irregular. Complaint was made of severe headache, as above. No trace of paralysis or disorder of sensibility; no nausea; mind remarkably clear; patient not anxious. Ophthalmoscope shows only somewhat enlarged retinal veins.

I agreed with Dr. Kinnicutt in diagnosticating a meningitis, probably of a tubercular nature; and I advanced the view that there was a lesion of the convolutions on the right side, in the neighborhood of the fissure of Sylvius, involving the excitable district of the cortex, and producing the localized epilepsy.

February 24th.—At 6 o'clock A.M., had a general convulsion, followed by vomiting; T. 37.1°, P. 108 and regular. At 2.30 P.M., T. 38.8°, P. 100; headache violent; vomited once. At 10 P.M., T. 38.5°, P. 100, regular; R. 32 and regular.

February 25th.—At 10.30 A.M., T. 39°, P. 100, regular. At 5 P.M., T. 38.5°, P. 100, regular. At 10 P.M., T. 37.7°, P. 100. Vomited twice during afternoon; no new symptoms; headache violent; urine albuminous.

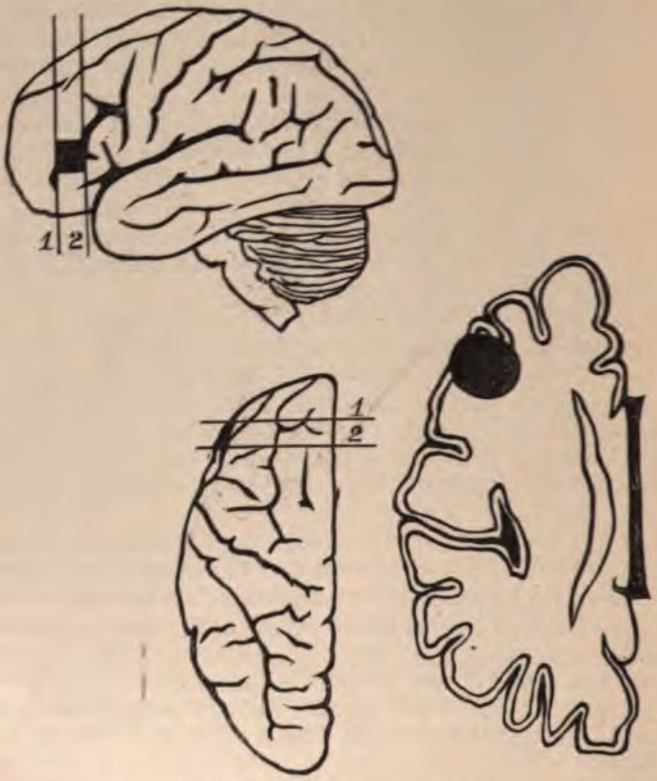
February 26th.—At 10 A.M., T. 39°. At 3.15 P.M., T 37.7°, P. 94, slightly irregular. At 10.30 P.M., T. 37.5°, P. 88, slightly irregular. Vomited once in twenty-four hours.

February 27th.—At 2.30 P.M., T. 37.5°, P. 80, R. 20. At 10 P.M., T. 38°, P. 88, irregular, R. 16–20, irregular.

February 28th.—At 5.30 A.M., pain in head intense; no motor disturbance of any kind; pupils normal and symmetrical; pulse and respiration irregular, the latter exhibiting the Cheyne-Stokes phenomenon typically. The mind is perfectly clear; strength good.

At 6 A.M., there occurred a general convulsion, followed by several others in rapid succession. There was loss of consciousness, and death took place quietly at 7.40 A. M.

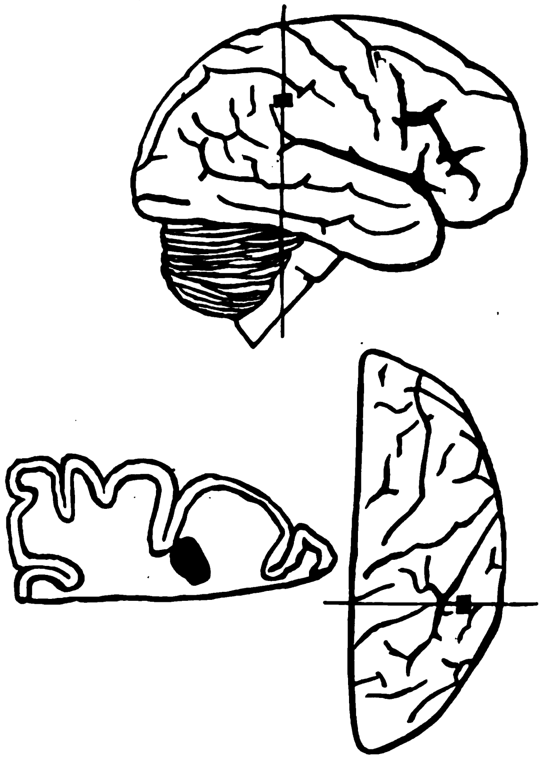
The treatment employed by Drs. Draper and Kinnicutt consisted in blisters to the neck, the giving of iodide of potassium, and, at last, to alleviate the severe cephalalgia, morphia hypodermically.



CASE VII.—ABSCESS IN LOWER PART OF SECOND FRONTAL CONVOLUTION, LEFT SIDE: NO SYMPTOMS,

I made the post-mortem examination at 3 o'clock P.M. on March 1st, in the presence of Drs. Draper and Kinnicutt. The condition of the left lung and remains of the pleura was one of great interest, but details on this point would here be out of place.

As regards the encephalon, we found a meningitis most marked at the base and sides of the brain, with purulent exudation in the meshes of the pia mater. There were no tubercles to be seen, and no marked lesion was to be found on the surface of the right hemisphere. On the left side, however, on the anterior lobe in front of the fissure of Sylvius, was a softened spot, probably an abscess. On carefully slicing the brain, a second lesion was found, however, and that in the right hemisphere just above the posterior extremity of the fissure of Sylvius. It is well worth while to state precisely the location and limits of these two localized lesions.



CASE VII.—ABSCESS IN RIGHT SECOND PARIETAL CONVOLUTION : CAUSE OF SPASM IN LEFT HAND AND FACE.

The first abscess, measuring 2 cent. in diameter, and lined by a soft membrane nearly 1 mm. thick, lay in the lower part of the second frontal convolution, and the anterior border of the third frontal convolution, on the left side, just in front of the speech-centre which has been referred to as injured in the cases of aphasia recorded in this essay. I am prepared to state, most positively, that the posterior part of the third convolution and island of Reil were perfectly healthy to the naked eye. The injured part is quite in front of Ferrier's centre No. 9.

During life, no symptoms, motor, sensory, or intellectual, occurred which might be connected with this large irritating and destructive lesion. Consequently, it seems to be fair to conclude that in some human brains the lower part of the second frontal convolution is not excitable, and contains no motor cell-groups.

As regards the second abscess, not larger than a pea, it was found wholly in the white substance just beneath the cortex of the anterior part of the second parietal convolution on the right side, just above the upper extremity of the fissure of Sylvius. This lesion, which, in all probability, gave rise to the spasm in the left face and hand, is placed just behind the parts which Ferrier considers to be centres for motions of the hand and wrist.

If any conclusion is to be drawn from a study of the second lesion, it is that in man fibres for the face and hand pass farther downward and backward in the hemisphere than would be indicated by experiments on dogs and apes.

A fact certainly well worthy of remark is, that a small lesion in an excitable district of the brain may produce well-marked symptoms, whereas a much larger lesion may exist in non-excitable regions without giving any sign of its presence.

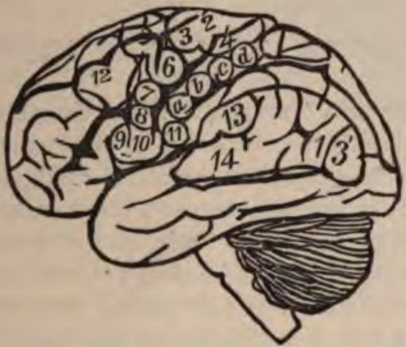
Conclusions.—The three cases in Part I. fully bear out Broca's (and Ferrier's) hypothesis of the existence of a speech-centre in the posterior part of the left third frontal convolution, and in the anterior folds of the island of Reil.

The case in Part II. is unfortunately worth very little; still it may serve to show that a lesion of the ascending frontal convolution may cause paralysis of the arm (and leg) without affecting the lingual and facial muscles, or producing aphasia.

The two cases in Part III. appear to indicate that spasm, limited to one side of the face and one upper extremity, may be set up by irritation of quite distant parts of the cerebral cortex, extending from region No. 5 to below region *d*, in Ferrier's chart. In this particular, these cases, while not contradicting experimental results, show that if there be such motor centres in man, they are not distributed quite as in monkeys and dogs.

The only case which appears to bear against the doctrine of localization of motor functions in the cerebral cortex is Case I. In this, it will be remembered, great recovery of voluntary power occurred, although a large part of the cortex of one hemisphere, including regions 6, 7, 11, *a*, *b*, and *c*, of Ferrier's chart, or, in other words, the so-called psycho-motor centres for the hand and forearm, the angle of the mouth and platysma, and those for the hand and wrist, were irreparably injured, nay destroyed.

I append a reproduction of Ferrier's chart of psycho-motor centres to render easy the determination of the injured parts in the above cases.



FERRIER'S CHART—1, Centre for Leg; 2, 3, 4, for Arms and Legs; 5, Extension of Arms and Hand; 6, for Biceps of Arm; 7, 8, for Angle of Mouth; 9, 10, for Lips and Tongue (Broca's Centre); 11, for Platysma; 12, for Head, Eyes, and Pupils; *a*, *b*, *c*, *d* (Ascending Frontal Convolution), for Hand and Wrist; 13, 13′, 14, Centres for Vision and Hearing.

The problem of localization of functions in the cerebral cortex is a grand one, and well worthy of thorough study. I am not prepared to accept the hypothesis as enunciated by Ferrier, yet it seems to me in the highest degree rash and unscientific to call it baseless and worthless, because a few recent negative cases can be brought forward against it. As for old cases bearing on these questions, I wholly reject them as cracked or weak links in a chain that must be made only of approximately perfect joints. It is surely the duty of all medical men to publish cases negatively or positively bearing on this question, but only when their observations have been made in an exact manner.

With a large accumulation of precise autopsies, with careful experiments by physiologists, with more embryological knowledge, and with unbiased comparative anatomy, some one may, in a few years, study and solve the problem.

THE ABUSE AND USE OF BROMIDES.*

Mr. President and Gentlemen: The time allowed by custom for the reading of a paper before a medical society will not permit me to treat the subject of the abuse and use of bromides as fully as its importance deserves. I shall only be able to consider the salient points of the topic, almost restricting myself to what I have observed in this branch of therapeutics.

The paper will consist of two parts. The first devoted to a study of bromism, or intoxication by the bromides; the second to a succinct statement of my own method of using the bromic salts in the treatment of epilepsy and other neuroses.

Bromine (from *βρῶμος*, a bad smell,) was discovered in 1826 by a French chemist, Balard, and to him we also owe the production of the bromide of potassium.

Bromide of potassium appears to have been soon tried by physicians, but it was not until 1840 that its physiological and true therapeutical effects were first apprehended. This was by a German, Otto Graf.†

In 1850 Huette,‡ of Paris, read to the Société de Biologie a remarkable paper, which may be considered as the basis of the modern use of the bromides. Huette observed and accurately described the general sedative effect of the bromides, their depressing action upon the sexual organs, the anæsthesia of the palate and throat, the mental torpor, the disorders of mobility, and the cutaneous anæsthesia produced by the drug. He also determined (against a number of physicians) its uselessness in late syphilis. Huette may, consequently, be rightly considered as having been the first to describe mildbromism.

With respect to the use of bromides in neuroses, and especially in hysteria and epilepsy, it is generally admitted that Sir

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* From the Journal of Mental and Nervous Disease, July, 1877, vol. ii. Read before the New York Medical Journal Association, April 25, 1877.

† De Kali bromati efficacitate interna experimentis illustrata. Lipsiæ, 1840. (From Voisin's Essay.)

‡ Recherches sur les propriétés physiologiques et thérapeutiques de bromure de potassium. Mém. de la Société de Biologie. 1850, vol. ii., p. 19.

Charles Laycock[*] was its originator and advocate, and that Brown-Séquard did the most to systematize and render successful the bromic treatment of epilepsy. For further historical considerations I would refer to Auguste Voisin's excellent essay published in 1875.[†]

From this time (1857) the bromides have been used by an increasing number of physicians in an almost endless list of diseases and symptoms. Among these may be mentioned Hysteria, Epilepsy, Infantile Convulsions, Puerperal Convulsions, Sexual Excitement, Chorea, Tetanus, Delirium Tremens, Insanity of active form, Melancholia, Cerebral Excitement and Insomnia, Somnambulism, Vomiting, Headache, Diabetes, etc., etc.

This general use of the various bromides (of potassium, sodium, ammonium, lithium, camphor, etc.) was largely empirical, the medicine being prescribed because of its quieting effects, and without strict regard to its physiological action.

From 1867 to the present time numerous researches upon the effects of bromides upon the healthy organism have been made by competent observers in various countries,[‡] and since the publication of these papers there has been, I believe, a more rational and moderate use of bromic salts.

The most important conclusions reached by these physiologists respecting the *modus operandi* of the bromides, are two in number. According to some (Brown-Séquard, Amory), the bromides act by causing contraction of arterioles and consequent diminution in the amount of blood in the nervous centres; while according to others (Eulenburg and Gutmann, Laborde, Wood), they affect the nervous tissues directly. All agree, however, in considering the physiological result of the action of the bromides to be lessened irritability of the nervous centres, especially in the motor tract.

I cannot, of course, now enter upon a discussion of this question, which is really only one phase or face of one of the greatest questions in medical philosophy, viz., whether biological processes are more dependent upon vascular (vaso-motor and hæmic) changes, or upon varying degrees of cellular activity, but I may be allowed to give it as my opinion that the bromides act mainly in the second way referred to, viz., by an action upon the anatomical elements (ganglion cells, chiefly) of the central nervous system. This belief is based upon physiological experiments in animals, clinical observations in man, and largely, also, by the phenomena of bromism; which last are, it seems to me, quite inexplicable by the first or vascular theory of the action of bromides.

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[*] The *Lancet.* 1857, i., p. 528.

[†] De l'emploi de bromure de potassium dans les maladies nerveuses. Paris, 1875.

[‡] For *résumé* of physiological action consult H. C. Wood's Treatise on Therapeutics. Philadelphia, 1874, p. 278, et seq.

Chiefly, in consequence of the prevalence of the empirical notion that the bromides are called for whenever there is excitement, and partly, also, because of the extreme application of certain theoretical views concerning the physiological and pathological importance of changes in the amount of blood in the brain and spinal cord, there has been, and is still, I believe, a great abuse or overuse of the various bromides, and it is not seldom that we meet with patients who have been kept in a condition of impaired nutrition and nervous atony for months or years, by means of these medicines, and with others (less numerous) who present the toxic symptoms of the drugs, who have bromism, so-called.

The remarks which follow upon the abuse of the bromides, are naturally divisible into three sections: 1, concerning the general description of mild and of severe bromism; 2, respecting the complication which bromism may cause in diagnosis; and 3, with reference to the legal aspects of bromism.

I. Bromism of varying degrees.—In a number of cases I have observed the following symptoms superadded to legitimate symptoms of disease: general debility, with weak pulse and coldness of the extremities; a tendency to stupor; slight difficulty in speaking, partly due to an aphasiform state; the bromic breath and acne. These persons were weak, anæmic individuals, who had been given the bromides for the relief of certain head symptoms, which were quite gratuitously supposed to be due to cerebral congestion. In some of these cases moderate doses of the drug had been taken for long periods of time, with frequent temporary relief to some symptoms. Yet all the while the patient's general condition had been kept below par, in spite of tonics and selected food. I have observed the same mild bromism, without any real improvement, in some cases of hysteria and hystero-epilepsy. Again, in melancholia, a disease in which cerebral nutrition is quite surely lowered and perverted, I have known injurious effects follow the prolonged use of bromides. In addition to the instances enumerated, there is a large class of patients who, without having any definite disease, suffer from nervousness, imperfect sleep, queer sensations about the head, and who constantly over-estimate their symptoms, and to whom the physician or druggist says, in an off-hand manner, "take a little bromide."

It may be said that often the giving of the bromides in the above manner does not produce positive ill effects ; but to this I would reply, first, that from what we know of the physiological effects of the bromides, such dosing must produce a general depression, or lowering of vitality, which few patients can tolerate ; and, second, that, on general principles, physicians are in duty bound to give no superfluous or non-indicated drug to their patients.

Bromism may be much more severe than depicted in the above statement ; it may attain the dignity of a distinct morbid state, with a clear symptomatology, a well-known course, and I am disposed to think, a central lesion. Huette, in 1850, gave a partial picture of this severe intoxication, and Prof. William A. Hammond has furnished us with a fuller representation of all its graver details.*

The chief symptoms of this condition are :

Cerebral : there is a gradually increased stupor, and dullness of intellect ; language is impaired ; failure of memory and difficult articulation ; the memory is much weakened generally, hallucinations, delusions, and even delirium may supervene.

Spinal : general debility becomes marked paresis, and a staggering gait, like that of an intoxicated person, is developed ; the facial and other muscles are tremulous ; the reflex functions of the palate and throat are abolished ; general cutaneous and mucous sensibility is much dulled ; the pupils are wide and sluggish ; the facial expression idiotic or maniacal ; the menses reduced or arrested ; the virile power reduced, etc.

Vaso-motor and trophic : the heart beats feebly ; the arteries carry less blood and show less impulse ; the peripheral circulation is sluggish and the extremities cold ; the breath is foul and quite characteristic ; the skin of the face and body is covered with acne ; the skin and mucous membranes are dry ; the saliva scanty and sticky; sometimes ulcers or a rupia-like eruption shows itself on the extremities.

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* On some of the effects of the bromide of potassium when administered in large doses. Quarterly Jour. of Psychological Medicine, iii., 1869, p. 46.

These symptoms may be so aggravated as to simulate dementia, mania, or general paralysis of the insane; and even death may ensue from extreme debility.

I desire particularly to insist upon the resemblance between bromism and general paralysis of the insane. In both we observe tremor of the facial and lingual muscles, producing a peculiar vibratory speech; in both there is an uncertainty in the performance of certain movements, as walking or using the hands for fine work; in both there is a failure of intellectual force and of memory. Even somewhat exalted notions may be present in bromism, though this is rare. In general paralysis we have other important symptoms, such as contraction and inequality of the pupil, sexual excitement, peculiar epileptiform seizures, remarkable remissions in the symptoms, and often good physical health, with tense arteries; all these symptoms being wanting in bromic intoxication. Severe bromism is, I am happy to say, very seldom produced, except during the early stage of the treatment of obstinate epilepsy, chiefly for the reason that the doses given for other affections are insufficient to bring about such a result, in the majority of persons. Dr. Hammond believes that bromism is rarely produced by doses less than 2.–3. of the bromide of potassium daily, and my experience would lead me to a similar estimate.

Occasionally we deliberately produce severe bromic intoxication. This is done in some severe cases of epilepsy, though even in these we seldom go farther than creating a state intermediate between the two conditions I have described. Again, bromism has been proposed as a cure for the morphine habit, or mania. Dr. Geo. M. Schweig,* of New York, has published a most interesting case in illustration of this procedure. The medication is certainly very heroic; but it is doubtful if any treatment not imminently dangerous to life, is not acceptable in such a terrible malady as the opium habit. Dr. Schweig's paper is, furthermore, an admirable study of the severe effects of the bromides.

II. Bromism as a complication in diagnosis.

The following case is related by Voisin.†

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* Cure of the Morphine Habit. New York *Medical Journal*, May, 1876, p. 495.

† Op cit. p. 68.

A patient who had been under treatment for epilepsy became, as his physicians thought, insane, and was sent to Paris to consult Voisin. The patient was found at a hotel in a state of violent mania, beset by frightful hallucinations of hearing, and shouting loudly. Later there was stupor, loss of memory, of affection, and of appetite; the walk was oscillating, and all movements were irregularly performed. Titillation of the nares and throat showed complete loss of reflex action, the hands were tremulous, and the facial and lingual muscles were the seat of fibrillary contractions. The pupils were equal, and the symptoms had developed very acutely after the use, during some months, of potassium bromide in doses of 6.-8. The medical officers of an asylum in which Voisin placed this patient thought he was mistaken in his diagnosis of bromism, and in his favorable prognosis; they looked upon the case as one of general paralysis of the insane.

In a week after the cessation of the bromides and the use of vapor baths, purging, black coffee, etc., the symptoms subsided, and in thirteen days the patient was sent back to his home in the country, quite well.

In a case which I saw in consultation, the addition of bromism to other symptoms led to the diagnosis of cerebral lesion of the gravest kind, when really only the basal dura mater was involved. The following is a summary of this to me instructive example of the evils of the purposeless giving of the bromides in large doses.

I was sent for to see Mrs. X. in a country town, near New York, on October 18th, 1875. She was under the care of a very intelligent practitioner, and had been seen by a prominent oculist of New York. I learned that this lady, then aged fifty-five years, had enjoyed good health during her adult life. In 1863, in Europe, she had an attack of mydriasis on the right side, without diplopia, or ptosis, or lesion of the fundus, or headache. This disappeared in three or four months. In 1870, having been well during the interval, while again in Europe, experienced internal strabismus of right eye with diplopia, but no pain. An oculist of Naples performed tenotomy of the internal rectus without relief. She had not then (and has never had) neuralgic pains in the legs, osteoscopic pains, sore eyes, or sore throat; never was dizzy or faint. In 1872 had pain in the head for the first time, in the shape of neuralgia of the right supra-orbital nerve. This pain has been present ever since with great variation in intensity. Patient was often awakened by severe pain at 3 or 4 o'clock A.M. The pain soon affected the whole of the right temporo-frontal region, with some extension into the eyeball and orbit. About one year ago (1874), and often since, had tingling in all the superficial branches of the right trigeminus. No irritation of the acoustic nerve. During the past year the sight of the right eye gradually failed, and slight exophthalmus appeared. Returned to America about one month ago, and was fairly well on board ship. Soon after landing, the local head and brow pains became much more severe, the pain apparently shooting through the right anterior lobe of the brain. There was no ptosis, but the eyeball was fixed in internal strabismus, without dilatation of the pupil. Bromide of potassium was then given internally in doses of 4. and 6. per diem, blisters were applied behind the ears, and morphia exhibited. Patient became weaker. On Oct. 1st, ptosis appeared, there was only perception of light in the right eye; vision normal in the left eye. The bromide of sodium was then substituted for the potassium salt, and given in doses ranging from 6. to 12. per diem. Patient grew weaker and weaker, was stupid, used wrong words, staggered while standing or walking; hands were tremulous. Oct. 12, bromides stopped and the iodide of potassium given in .60 doses three times a day. About this time slight anæsthesia of the right brow was discovered.

I found the patient, on the 18th, in a state of hebetude, speaking a little thickly and slowly, and quite often using the wrong word. She is perfectly intelligent. There is an abundant flow of buccal saliva and nasal mucus, but no acne. The left side of the face and the tongue are normal. Smells with both nostrils. On the right side there is ptosis, and on raising the eyelid the eyeball is found immovable nearly in the median line, its pupil of medium size and fixed; only perception of light on this side. The ophthalmoscope shows simple atrophy of the optic nerve; there is no choking of the disk and no trace of hemorrhages in the retina. The fundus of the left eye is normal, and its vision is good; field not impaired. The seat of pain is as described above. The brow, temple, and fronto-parietal region on the right side are partly anæsthetic. There is no palsy of the face or extremities, no anæsthesia of fingers, no referred sensations (numbness, etc.). The walk is titubating but not hemiplegic. Patient repeats that she has never lost consciousness. Her pulse is regular, beating 80 in the minute, and her buccal temperature is 37° C. The attending physician and the consulting oculist had concurred in diagnosticating a tumor in the right middle fossa of the skull, involving the brain.

My own conclusion was that we had to deal with an inflammatory affection of the dura mater in the right middle fossa of the skull, compressing the nerves, etc., which pass through the optic and anterior lacerated foramina, and not involving or affecting the brain substance. The cerebral symptoms present seemed to me to be those of bromism, partly by their intrinsic characters and mode of appearance, and partly because they were not those which a lesion at the base of the brain, on the right side, could produce. Furthermore, I rejected the idea of a cerebral lesion because of the absence of hemiopia and of lesion in each eye, of hemiplegia on the opposite side, both of which symptoms a tumor in the middle fossa must of necessity produce by pressure upon (*a*) the right optic tract and (*b*) the right crus cerebri.

As to the nature of the inflammation, I gave no opinion; the social position of the patient, her blooming family of children, and her own medical history previous to 1870, being opposed to a syphiltic theory. Still I considered that we were in duty bound to give her the benefit of the doubt, and I urged the attending physician to continue withholding the bromides, to give the iodide of potassium in gradually increasing doses, to relieve the pain by hypodermic injections of morphia, and to support the patient with food and stimulants. It will suffice, for the present purpose, if I state that after 15. of iodide of potassium per diem was reached and passed, improvement began and progressed rapidly. The medicine was carried up to 24. a day, and held at that dose for some time, then gradually decreased; doses of .60 being taken as late as the spring of 1876. The symptoms of supposed cerebral lesion passed away in a few days, and the local symptoms gradually disappeared, except the atrophy of the optic nerve. I met this lady a few months ago, and she seemed in perfect health, with the exception of slight imperfections in the movements of the right eyelid and eye-ball, and of loss of vision in the eye.

It would be easy for me to relate other cases, illustrating the proposition that bromism may embarrass diagnosis, but my space is limited, and the two examples given above are perhaps sufficiently demonstrated.

I should add, however, that apart from the above special symptom-groups, the use or abuse of bromides may give rise to a condition of general debility, and to a weakness of the heart, which are not then by any means as serious as when not produced by the bromides.

III. Bromism in its medico-legal aspects.

I am not aware that bromism has ever been brought into the courts as a matter for study and decision, but it may eventually be so under several circumstances.

First, with respect to the responsibility of the physician administering the medicine which so debilitates a patient, physically and mentally, as to expose him to various mishaps. For example, I know of a case in which the patient, suffering from acute bromism, fell asleep in a railway station, and was robbed of four hundred dollars, so great was the stupor produced by bromides, by thieves who undoubtedly wondered at the man's indifference to their manipulations. Dr. Hammond * relates, in his essay on Bromism, the case of a gentleman, one of his patients, who was arrested on the street for drunkenness, and locked up over night in spite of the doctor's remonstrances and explanation.

Second, as to the patient's responsibility for criminal acts committed while brominized. It is perfectly possible that such a patient shall take from a store articles not paid for, through defective memory; that he shall be mistaken in the identity of persons, and thus be led to be improperly familiar or abusive; or that he shall enter a house or room not his own, etc.

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* *Journal of Psychological Med.*, l. c.

Third, with respect to the legal capacity, both for ordinary business and for testamentary disposition, of bromized persons. Each case will, of course, have to be studied by itself, but it must be admitted that in some cases of bromism the stupor, loss of memory, and aphasiform difficulty are so great that the patient is, for the time being, as truly non compos mentis as if he had a natural secondary dementia.

A decision will be the more difficult to reach, because in mild and in moderately severe bromism the judgment and general intellection are remarkably well preserved, behind a veil of striking superficial symptoms, as impaired articulation, stupid expression, staggering gait, partial weakness of memory, muscular weakness and tremor, etc. Again, in some cases, it will be necessary to make a close analysis of the patient's antecedents, in order to clearly ascertain how much of the mental impairment depends upon the medicine, and how much upon the disease for which the medicine was prescribed or taken.

Fourth, with reference to the production of death through bromism. This idea is suggested by reading a case of fatal bromism related by Clarke and Amory,* in which a nurse, literally applying orders given him by a physician, continued giving enormous doses of bromide of potassium in spite of progressive weakness. When seen by Dr. Clarke, the patient was past recovery, and sank in a week.

There is a possibility that this procedure may some day be repeated with criminal intentions; *e.g.*, for the purpose of getting rid of a burdensome and incurable invalid.

I shall now proceed with the second part of this paper, viz., a statement of my own mode of using the bromides in the treatment of epilepsy and other neuroses.

In prescribing the bromides for epilepsy, I have been guided by ideas which can, perhaps, be best expressed in the form of terse propositions.

1. In view of what we know of the physiological and toxic effects of the bromides, and in accordance with either of the two generally received hypotheses of their modus operandi, anæmia and debility, or congenital feebleness, contra-indicate prolonged use of the bromides.

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* The Physiological and Pathological Actions of the Bromide of Potassium, Boston, 1872, p. 62.

2. The bromides are, on the contrary, well borne by persons of fairly full habit and good nervous power.

3. The bromides are indicated in cases of abnormally great irritability of the nervous system, in its motor (muscular and vaso-motor) and ideational tracts.

4. Epilepsy is so serious a disease, one which, if not interrupted, kills the patient, or reduces him to dementia, that we are justified in using unusual and heroic measures in its treatment. Hence, the contra-indications named above are to be much less regarded in the management of this formidable neurosis.

5. As a corollary to the last proposition, I may state that I consider epilepsy to be the only disease for the cure of which we are justified in deliberately producing a degree of bromism.

My method of prescribing the bromides in a common case of "idiopathic" epilepsy, is the following:

I employ one of two solutions,* made according to a standard formula.

℞	Potassii bromidi	30.
	Ammon. bromidi	15.
	Aquæ font.	220. cc.

M.

S. To be given by the teaspoonful.

℞	Sodii bromidi	30.
	Ammon. bromidi	15.
	Aquæ font.	220.

M.

S. To be given by the teaspoonful.

These simple solutions, which I have found much more palatable to most patients than those made with infusions or syrups, contain forty-nine doses; *i.e.*, each teaspoonful contains .60 of the potassium or sodium bromide, and .30 of the ammonium bromide.

The solution is given several times a day, nearly always so divided as to give by far the largest dose in the evening. This is Brown-Séquard's rule, and the principle involved is to keep the system thoroughly under the influence of the drug during the night.

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* Latterly chloral hydrate has been very frequently substituted for the bromide of ammonium.—[R. W. A.]

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I direct for an adult male epileptic, that a teaspoonful shall be taken before each meal, and two teaspoonfuls at bed-time, largely diluted. In the case of delicate males and of females, I at first prescribe only a teaspoonful before two meals, and two teaspoonfuls at bedtime, and in some young persons or very small and tender adults only, one dose before breakfast, and then at bedtime. The patient taking these initial or trial doses is carefully observed, the sensibility of the palate and throat frequently studied, and information obtained from the patient and his friends as to the absence or presence of stupor. Guided by these signs, or their absence, I cautiously increase the bromide, still keeping the nocturnal dose the largest, until slight bromism is produced, as evidenced by absence of reflex movements in the throat, and slight stupor. I pay but little attention to acne. During the rest of the treatment, I aim to give the patient just as little bromide as shall prevent attacks of epilepsy, yet I nearly always find it necessary to keep up slight bromism for months.

The precise amount required per diem in a given case can only be determined by careful observation of that case, and is not to be deduced from general experience. At times, remarkable idiosyncrasies are observed which inexplicably render the patient very susceptible or very rebellious to the bromic influence. Very many of my patients take, month after month, one dose (1. of the two bromides) before each meal, and three doses (3. at bedtime); a total of 6. As extremes illustrating peculiarities, I may cite the case of a girl of twelve years, who for weeks took 9. per diem without bromism, and that of a young lady of twenty, who was decidedly influenced by one teaspoonful before breakfast and two at bedtime; a total of only 3. per diem. In the latter case, had I given the usual doses taken by adults, I should have produced severe bromism.

With respect to children I find that they tolerate the bromides (and iodides as well) in relatively large doses, and little patients of mine often take 4. of the bromides a day; while to mere infants I give (after careful trial of smaller doses) 1.2 to 2.5 a day.

It appears to me very important to thoroughly dilute the bromides, in order to facilitate their absorption; I usually direct that the dose be taken in a wineglassful or half a tumblerful of water. Furthermore, I give the medicine on an empty stomach.

With respect to the practice of giving a very large dose at bedtime. Theoretically, upon physiological grounds, it appears right to obtain the greatest bromic action in those hours when the reflex power of the motor part is probably heightened, and when epileptic seizures often occur; and again, as a great number of hours must elapse before another dose can be taken, it seems right to give an extra large amount to keep up the medicinal effect. Empirically there can be no doubt of the great importance of this rule. Brown-Séquard's extraordinary success in the treatment of epilepsy was in part due to this, and I have several times seen patients who had been taking a large amount of the bromides in three equal doses without much improvement, who have had fewer attacks immediately after subdividing the same amount in such a way as to give a large dose at bedtime. For example, 2. three times a day did a little good, but 1. before each meal and 3. at bedtime checked the disease much more.

Another of the reasons of Brown-Séquard's success was his positive direction that under no circumstances should the bromides be discontinued; and I have always studiously followed his teachings in this matter. The bromides may be *diminished* but never *stopped* until the word *cure* can be pronounced. Even during intercurrent acute diseases, as colds, fevers, accidents, the bromides should be given regularly, though in reduced doses, partly because the nervous system resists less in that condition, and partly because such attacks of illness or accident interrupt the epileptic habit. The omission of the bromides for a very few days may allow a fit to occur, and thus destroy the good work done by months of patient care.

How long must the bromides be taken in epilepsy? This is a question to which we can as yet give no answer. Brown-Séquard and Voisin give it for at least three years *after the last attack*, and I think that this is a minimum of time. I have twice been grievously disappointed by the return of attacks after an immunity of over two years, and others have known recurrence to take place after even a longer period.

Some patients who have had epilepsy for many years are partially demented, and take the bromide unkindly; they become irritable, feeble, and have nearly as many attacks as without the drug. In such cases parents will often ask you if it is worth while to give the bromides systematically, and to bear with the bromic symptoms. I generally answer this question negatively, yet state to the parents that as the patient may die in a paroxysm it is our duty, on general principles, to do anything which shall diminish that risk.

It will be inferred from the foregoing that I rely upon the bromides of ammonium, sodium, and potassium for the treatment of epilepsy, and this is in one sense so.

No medicine, it is now generally admitted, has such power over the epileptic habit, and does good in so many cases, as the bromides, and it would seem as if the day for trifling with such doubtfully efficacious medicines as zinc oxide and sulphate, copper sulphate, belladonna, strychnia, setons, diet, etc., had passed away. With the bromides of calcium, lithium, zinc, and arsenic, I have had little or no experience. The last named is loudly vaunted by Clemens, of Frankfort on the Rhine.

I would not, however, have it understood that I employ *only* the bromides in the treatment of epilepsy. On the contrary, what measure of success I have is owing in part to the fact that I made a large use of other means, together with the bromides; and this seems to me so important that I shall take the liberty of digressing a little to specify what this adjunct treatment is.

In the first place, I employ means which tend to counteract the unpleasant effects of the bromides.

The acne may to a certain extent be prevented by administering arsenic from time to time, either in the shape of the solution of arsenite of potassa, or of arsenious acid. Sulphur ointments, mercurial plaster, alkaline lotions, may also be employed.

The general debility or slight paresis produced by the "continuous dose" (Clarke) of bromides is corrected by strychnia, by nux vomica and zinc oxide, and by quinia. Drowsiness and the more serious symptoms of bromism are relieved by inhalation of nitrite of amyl, by stimulants, and by quinia. The anæmia and general depression of the vital functions produced in the course of the management of a case of epilepsy, I meet by care in giving the patient nutritious diet, by giving cream or cod-liver oil, and by administering such medicines as iron, quinia, phosphorus, strychnia, with nitro-muriatic acid, wine, beer, or whisky, and by regulating the patient's hygiene.

Important hygienic rules in the treatment of epilepsy are the avoiding of large meals at night, regulating the functions of the bowels, kidneys, and skin, early rising, and great moderation in sexual gratification.

In the second place, I employ, in some cases, a few medicines which act more directly upon the morbid state of the nervous centres. These are belladonna, cannabis indica, oxide of zinc, strychnia, sulphates of zinc and copper, etc. My favorite is the first named, and I have known the best effects to follow its association with the bromides. For example, a patient passed into my hands after having been a long time under the treatment of a distinguished physician with a moderately good result; under a given quantity of the bromides she had attacks about fortnightly. I did not increase the bromides or change the method of taking them, but at once gave extract of belladonna in doses of .02 three times a day. The patient acquired a dryness of the throat, and the attacks were reduced in frequency to once a month, once in two months, three months, and she has now been thirteen months without an attack. Of course the belladonna was not continued during all the two years of treatment. At first it was used for two or three months in succession, and afterward given from time to time.

During the many months or years of the treatment of a case of common epilepsy, I ring the changes on the medicines above enumerated, nearly always giving something in addition to the bromides. And I may be allowed to repeat that the bromides, though often changed in amount, and sometimes in kind, are never withheld.

The treatment of cases of epilepsy in which a definite causative lesion can be made out, is, of course, somewhat different. I refer now to epilepsy due to syphilitic lesions, to peripheral disease, to cranial and neural injury, etc. In these varieties I use the bromides to combat the epileptic habit, to prevent discharges (using Hughling Jackson's phraseology), and at the same time meet the special indication by using mercury and iodide of potassium, by correcting visceral disease, by removing some external irritating cause, or by an operation like trephining, neurectomy, etc.

In other neuroses, I have used the bromides sparingly, and never continuously.

Hystero-epilepsy and hysteria have not seemed to me much benefited by the bromides. As far back as 1857, Laycock remarked that the bromide of potassium was especially successful in hysteria of distinctly ovarian or uterine origin. I would not condemn the use of the bromides in hysteria, but would protest against their being given in such a manner as to produce bromism.

Insomnia, I think, is often treated by bromides, upon the purely hypothetical indication of causing anæmia of the brain—an indication reposing upon insufficient physiological experimentation, and upon belief in the notion that the bromides directly produce cerebral anæmia. Many cases so viewed might be much more quickly relieved by chloral, or by a glass of ale, or by correcting indigestion. A case of well-marked insomnia needs, it seems to me, to be investigated in the broadest manner, without failing to keep in mind that this symptom may depend upon a number of pathological conditions. As to the immediate cause of sleep, I believe it to be due partly to the waste of tissues generally, and the presence in the blood of an accumulation of the products of retrograde metamorphosis (Preyer's theory), and partly to the exhaustion of the cerebral tissue itself. The anæmia which is observed in the brain during sleep is, it appears to me, a concomitant, or consequent phenomenon, in obedience to the general law that a tissue in repose contains less blood than one in action.

Insanity is often, I believe, erroneously treated with the bromides. I have several times seen patients with melancholia made weak and wretched by large doses of the bromides which failed to make them sleep; and in mania I have known precious time wasted in vain attempts to get sleep by these medicines. I would only employ the bromides in insanity to meet a few indications, such as a tendency to epileptiform attacks, or abnormal sexual excitement, or great nervousness not caused by delusions.

I have not used the bromides in chorea and neuralgia. It is vaunted in migraine, but in some half dozen cases in my practice it has given absolutely no relief to the attack. Extreme irritability of the bladder with pain, in a female, was very greatly relieved by a retained vaginal injection of 2. bromide of ammonium. The idea was to produce anæsthesia of the vagina and vulva. Upon a similar principle we all use gargles of bromides in neuralgic or myalgic sore-throats, and in the cough of laryngeal irritation, with fair success. Vomiting in pregnancy, after morphia, ether, etc., may be arrested by judicious use of the bromides. It has been proposed to give bromide of potassium an hour or so before administering ether, or giving opium or morphia, with the view of preventing nausea and vomiting. This practice has seemed successful in my hands also.

Hay-asthma, or hay-cold, is a disease for which multitudes of medicines have been tried, without much good result. Last year, I induced two or three persons suffering from this disease to employ a strong gargle of bromide of ammonium, and to wash out the nasal passages with a weak solution of the same salt several times a day during the attack. The result was so gratifying that I am disposed to ask physicians to give a fair trial to this treatment during the coming summer and autumn. The gargle to be of the strength of 4. or 8. of bromide to 30. cc. water; the solution for the nares, much weaker, of from .60–2. of bromide in 30. cc. water.*

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* New York *Medical Record*, Nov. 11, 1876, p. 737.

A CONTRIBUTION TO THE THERAPEUTICS OF MIGRAINE.*

GENTLEMEN:—The contribution to the therapeutics of migraine which I have the honor to read this evening, will probably strike you as very fragmentary and inconclusive, but I would ask you to consider in a charitable spirit that it is the result of only a few hours' work, and that it is intended as a suggestive rather than a didactic and formal essay.

So short has been the time which has elapsed since I was asked to participate in this evening's work, that I have not been able to collect scattered notes of cases and to make inquiry of former patients; both of which would have been necessary had I wished to base my statements upon statistics. At some future time it may be possible to supply the data upon which the succeeding assertions rest.

Briefly stated, my thesis is THAT BY THE LONG-CONTINUED USE OF CANNABIS INDICA, MIGRAINE OR SICK-HEADACHE MAY BE CURED, MUCH RELIEVED, OR MITIGATED IN SEVERITY.

This idea is not by any means original with me, but was brought out by an English physician, Dr. Richard Greene, who published a short article upon the subject in *The Practitioner*, vol. ix., p. 267, London, 1872. After reading the article I immediately began using the remedy, cannabis indica, as directed by Dr. Greene, and have continued to do so ever since. My former partner, Prof. William H. Draper, has also used the treatment somewhat during the same period of time; and both of us have been much gratified by the results obtained. I may add that some inquiry has convinced me that, in this country at least, the article passed unnoticed, and the plan has not been generally tried.

Before proceeding to give details concerning the treatment, it might not be amiss to recapitulate the diagnostic characters of migraine or sick-headache. This affection is essentially neuralgic in its chief manifestations, viz., a severe or excruciating pain in the head and orbit, but not along the superficial branches of the trigeminus. It affects both sexes, from the age of six or ten years to that of forty or fifty. In some patients it makes its first appearance at puberty and terminates before the sixtieth year. In females it may, after undergoing aggravation or transformation, cease at the menopause. Very rarely does the disease cease before thirty, and still more rarely does it first appear at that age.

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* Read before the Section on Practice of Medicine in the New York Academy of Medicine, Nov. 20, 1877. Reprinted from the N. Y. *Medical Record*, Dec. 8, 1877.

Migraine is pre-eminently an inherited disease, perhaps more directly so than any other neurosis. I possess numerous tables of families in which many members of three generations were affected.

Migraine is periodic in its manifestations, nearly as much so as epilepsy; patients have attacks every two months, or monthly, or every week—seldom several in a week. In some women the periodic return of migraine coincides with menstruation.

An attack of sick-headache usually begins in the very early morning and lasts all day—seldom longer in uncomplicated cases. In many cases certain premonitory symptoms precede the occurrence of pain. The day or evening before the attack some feel unusually bright and well. At the earliest waking on the day of attack there may be chilliness or numbness of a limited part of the body, dim vision, colored vision, or hemiopia. These optical disorders are of exceeding interest, and are best observed in those patients whose attacks begin some time after rising. They usually last less than half an hour. Although amblyopia, hemiopia, photopsia are often very serious symptoms, yet in migraine they lose their prognostic significance. In other persons nausea is an early symptom. Pain follows upon the above disturbances and sometimes makes its appearance without them. It is usually in one side of the head, hemicrania; deeply placed "in the brain" or "back of the eye," as patients tell us; it grows in intensity, is sharp, or beating, or pressing, and may reach such a degree of severity that patients strike their heads violently against hard objects, use chloroform, or beg for hypodermic injections of morphia to obtain relief. During the existence of this pain, which may extend to the rest of the head, there is hyperæsthesia of the eye and ear, great irritability, pallor of the face, cool skin, intense nausea, and severe vomiting. So prominent a symptom is vomiting, so early does it appear, and so abundant is the matter ejected, that the sufferers generally, and, I regret to say, physicians occasionally, consider the headache as caused by "biliousness," thus reversing the true order of cause and effect. For a while after vomiting there may be some relief to the suffering.

Toward evening the pain diminishes in intensity, changes its character to a dull general headache, and after a night's sleep the patient awakes quite well; in many cases feeling better than before the attack. Sometimes, however, in gouty subjects, or in women at the menopause, headache more or less typical will endure for two or three days.

It should be added that there are cases in which no nausea or vomiting appears; and patients are disposed to separate these from the category of sick-headache, and speak of them as "nervous headaches." I believe that these two varieties are of the same general kind—of the migraine type.

It would be out of place in this short paper to trace out the varieties and transformations of migraine, and I have only said enough of the symptomatology to make it unmistakably clear what are the cases in which the plan of treatment about to be presented is applicable.

The pathology of migraine is one of the most open questions in medicine, and I can only briefly state my own opinion, reached by a careful study of physiological considerations and clinical data. I believe with Anstie and many others, that a lesion (at present undemonstrable) exists or occurs in those parts of the pons and medulla oblongata which give origin to the sensory roots of the trigeminus. Various systemic states, and various irritations from the external world,* the abdominal organs, the cerebrum, serve to provoke the attacks.

One very potent exciting cause of attacks is mental overwork or anxiety; another generally recognized is that condition of the system in which oxalate of lime appears abundantly and frequently in the urine, and in which uric acid quickly separates from it—in brief, acidity, or a gouty disposition. Indigestion may also be an exciting cause.

Guided by the above pathological and ætiological notions, I have treated migraine by—

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* Of late Dr. Seguin was inclined to ascribe some migraines, especially bastard or non-typical attacks, to an optic or visual origin—to eye-strain.—[R. W. A.]

1. Treating the patient, and removing all exciting causes.
2. Treating the attacks themselves.
3. Treating the disease, or the supposed fundamental pathological state in the nervous system.

First.—The treatment of the patient consists in removing all relievable exciting causes, and more especially in correcting acidity. For this purpose I employ the ordinary means, viz., giving nitro-muriatic acid and alkalies, and greatly reducing the saccharine and amylaceous foods of the patient.* In cases attended by debility, anæmia, and imperfect nutrition, it may be necessary to resort to tonics, including cod-liver oil.

Second.—Treatment of the attack. The first thing to be done, in my opinion, is to place the patient under circumstances which secure quiet and semi-darkness. The attempt to "fight out" a sick-headache is nearly always vain, and may be injurious. It is better not to allow the patient any food, not even liquids, until toward the close of the attack, or even not till next day; by this, nothing is lost, and much wretchedness is avoided. Ice, or ice washed in brandy, is grateful.

If the patient have a warning (aura of migraine) before nausea or pain, much can, I believe, be done to cut short the attack or diminish its severity by the use of guarana, caffeine, or croton chloral hydrate. In my hands, guarana or the powder of the seeds of *paullinia sorbilis*, has proved very efficacious. I have prescribed the fluid extract of guarana, Caswell & Hazard's Elixir of Paullinia, the French Paullinia powders, and powdered guarana prepared by our druggists, and all of these preparations have in my hands often cut short or prevented attacks, *if given in the early stage of the disorder.*

Of the elixir or fluid extract I give a teaspoonful, to be repeated twice, at an interval of an hour. The powders are administered in 1.2–2. doses, also repeated every half hour or hour. I think that I may report that nearly one-half of my patients have derived great relief from some preparation of guarana, and that in several of them attacks have been absolutely prevented, and they have been enabled to go about on the same day.

Caffeine, in doses of .12, repeated every hour, until three or four doses have been taken, I have lately employed, upon the recommendation of my friend Dr. Geo. M. Beard, and it has appeared to do good.

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* Since writing this paper Dr. Seguin's belief in the lithæmic element in many cases of migraine, has been very much strengthened by the great success often attending this plan of treatment.—[R. W. A.]

Croton chloral hydrate, recently recommended in all neuralgic affections of the head and face, I have recently prescribed in doses of 1.-1.2 repeated every hour until four doses are taken or relief obtained. This remedy is to be used more especially in cases where pain is the first symptom, and in other cases if seen when the pain is fully established.

I have no personal experience with the use of large doses of bromide of potassium and of alcoholic stimulants, for the relief of attacks.

Hypodermic injections of morphia, .02-.03, and atropia, .001, have permanently relieved attacks in a few of my cases; but I am very reluctant to employ this means, so fraught with the danger of the formation of the opium habit. I never allow my patients to take opium or morphia themselves in this disease.

I would add that there is very probably a real ultimate usefulness in shortening or preventing every attack which may threaten to occur during the systematic treatment of the neurosis. We may thus be doing a good deal to interrupt the *morbid habit* which the nervous centres have acquired.

Third.—Treatment of the disease. No treatment of this sort had been tried, to my knowledge, before Dr. Greene made his remarkable researches upon the effect of cannabis indica. Dr. Greene reported cases of many years' standing as having been months and years without attacks while and after taking cannabis indica, and in other extremely bad cases marked reduction in the frequency and severity of the attacks was obtained.

I have said, in the opening page of this small contribution, that I and a few medical friends have used the cannabis treatment ever since Dr. Greene's publication, and with satisfactory results.

The principle of the treatment is to keep the nervous system steadily under a slight influence of cannabis for a long period of time; in other words, we are to employ the "continued dose" of the remedy, as Clarke and Amory say in speaking of the use of bromide of potassium in epilepsy.

I give to adult females .02 of the alcoholic extract of cannabis indica before each meal, increasing the dose after a few weeks to .03. Males can generally begin with .03, and it is well to give them .045 in two or three weeks. These doses must be taken with the greatest regularity, just as faithfully and regularly as bromides in epilepsy. Indeed, when beginning such treatment, I usually obtain a promise from the patient that he will regularly take the pills for a period of three months.

As a rule, no appreciable immediate effect is produced by the above doses, though I have known lightness of the head and slight confusion of mind to result from an initial dose of .03 three times a day.

Under this apparently and essentially simple plan of treatment, I have known what may be termed excellent results to be obtained. Of course, I do not mean to say that all my patients have been benefited, but, without a statistical table, so difficult to construct from the experience of private practice, I feel certain that about one-half of my cases have been relieved. A few—two or three—after being more than a year without return of their migraine, have passed from under immediate observation. One of these now very rarely has headache, although for several years he has taken no medicine. The majority of patients relieved have obtained months of freedom from attacks while taking the remedy.

I think that we may say of cannabis for migraine that it is nearly as efficacious as the bromides in epilepsy. Both *may* cure, both *do* bring about remarkable interruptions in the series of attacks, both must be employed in the shape of the continued dose.

Cannabis in migraine is less effectual than the bromides in epilepsy, but, on the other hand, it is superior to them in not producing unpleasant or injurious effects.

My friends and former partners, Drs. William H. Draper and Frank P. Kinnicutt, have used the above plan of treatment frequently in the last five years, and their results substantially agree with my own.

Some surprise naturally arises upon seeing so much good done by small doses of a neurotic medicine in a disease so deeply rooted as migraine. Our wonder may never cease respecting the *modus agendi* of the drug—its essential potent action; but its gross and practically interesting effect is very analogous to a well-established acquisition of empirical therapeutics. I refer to the successful employment of belladonna or atropia in epilepsy. This treatment, especially vaunted by

Trousseau, is by no means useless, although it is no longer fashionable since the more useful bromide treatment has come into general use. I still, however, employ belladonna in epilepsy in conjunction with the bromides, and this combination sometimes brings about gratifying results.

I may be allowed to briefly mention one illustrative case. When Dr. Brown-Séquard went to Europe in 1875 one of his patients came under my care. She had a bad form of epilepsy, and in spite of the most skillful use of the bromides by her illustrious physician she had been having a fit every two weeks for months. I made little change in the amount of bromides she was taking, merely substituting my own simpler solution for Brown-Séquard's mixture, and gave her .015 of belladonna three times a day—just enough to keep her throat a little dry. From the very beginning of treatment the epileptic attacks became fewer; intervals of one, three, and fourteen months being obtained. In the present year, owing to the uncontrollable cause of the epilepsy, she has had three or four seizures.

A close parallel may, I think, be drawn between the two diseases, epilepsy and migraine; and between the two remedies, belladonna and cannabis; thus, in my opinion, logically fortifying the propositions advanced upon empirical grounds, that cannabis is useful in the treatment of migraine.

1. Migraine and epilepsy are both nervous affections characterized by the occurrence of periodical attacks; the attacks themselves in both diseases are largely made up of vaso-motor disturbances; in both it is probable that the medulla oblongata is primarily or secondarily diseased; both affections occur in the same families, and may be present at successive times in the same patient. The late Dr. Anstie has expressed the opinion that the two diseases are akin, and states* that migraine may develope into genuine epilepsy. I have in my private case-books cases illustrating this proposition, and I am now treating a physician who states that after nocturnal epilepsy appeared, before beginning bromide treatment, his old migraine grew less frequent and less severe.

2. As regards the two remedies, cannabis and belladonna: both are intoxicants and deliriants; both dilate the pupil, and it is probable that the action of both upon the central nervous

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* The Practitioner, vol. ix., 1872, p. 356

system, when administered in the shape of the continued dose, is very similar.

In conclusion, I would earnestly ask the gentlemen who have honored me with their attention this evening, to give the cannabis treatment of true migraine a critical trial.

BULBAR PARALYSIS (AN ATYPICAL CASE OF LABIO-GLOSSO-PHARYNGEAL PARALYSIS).*

Male, aged 67. Patient of Dr. McCready, seen in consultation Sept. 25th, 1876. He was a steady drinker. Probably no syphilis. At least a year ago great difficulty in swallowing began, especially of liquids, and it steadily increased. In the winter of 1875–6, Dr. McCready was suddenly called and found patient in extreme and dangerous orthopnœa, without cardiac or pulmonary cause. The respiration was of the Cheyne-Stokes type, a few rapid acts of breathing, succeeded by a long pause, with shallow and short inspirations. Ever since, breathing has been more or less of this type. Children have noticed staggering gait and stooping in the last few months, and in the same period a degree of imperfection in articulation has shown itself. On Sept. 19th, partial right hemiplegia without loss of consciousness. Dr. McCready saw patient shortly afterward, and made sure that although the power of articulation was nearly abolished, there was no aphasia. After this attack the breathing became much more difficult, and deglutition has been nearly abolished; almost nothing being swallowed until to-night. Food has been administered per rectum. I found the patient in a state of semi-stupor, from which he could be roused. The pupils were normal; expression dull; lower part of face expressionless. Mouth hangs half open and its right corner and cheek drop. The breathing is shallow and rapid for a few seconds, then almost imperceptible (Cheyne-Stokes type). The grasp of both hands is weak, that of the right hand weaker. In attempting to talk patient makes great effort, but the sounds are almost inarticulate and guttural. He names daughters, days of the week, etc., and seems not to be at all aphasic. Labial sounds are best made, cannot distend cheeks with air or whistle, but purses lips fairly well. Tongue is only partly projected; it shows neither atrophy nor tremor. An autopsy showed uniform atrophy of cerebrum, sub-arachnoid effusion, *état criblé* in extreme degree. No other lesion appreciable to naked eye.

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* Journal of Nervous and Mental Diseases, vol. v., Jan., 1878, p. 134. Abstract of a paper read before the New York Neurological Society, Dec., 1877.

Under the microscope, lesions of the medulla oblongata usually present in this disease were found.

LOCALIZED BASAL MENINGITIS IN CHILDREN.*

GENTLEMEN:—I desire this afternoon to call your attention to a class of cases in which the use of the ophthalmoscope is strikingly advantageous, and this in the hands of those not *expert* in the handling of the instrument.

CASE I.—This little girl, aged six years, was brought to my class at the Manhattan Hospital a couple of weeks ago, with the following simple history: For two or three weeks she had complained of headache, had vomited frequently, and on February 9 (a week ago) internal strabismus appeared. The patient has not complained of impairment of vision, she has not had fever, spasm, or delirium. Constipation has, however, been marked. She is anæmic looking, a small brother of hers probably has phthisis, and one child of the same parents is said to have died of "brain fever." My assistant at the Manhattan Hospital, Dr. Adam, immediately examined the child's eyes with the ophthalmoscope, and found double neuro-retinitis, a diagnosis which I concurred in, and which was verified by Dr. Webster in the Ophthalmic Department of the Hospital. Consequently, the most important symptom was the one revealed to us by the use of the ophthalmoscope. I made the diagnosis of basal meningitis localized about the chiasm of the optic nerves, probably without tubercular deposit. The child was blistered behind the ears, and given .60 of potassium iodide three times a day, with instruction to increase the dose by .30 per dose, every second day.

As you see the child now she does not seem sick, and were it not for the convergent squint, you would probably consider her as only a delicate, anæmic child. In the last few days the headache and vomiting have ceased, and improvement has begun.

I shall now relate two analogous cases from my private practice.

CASE II.—Referred for examination to Prof. H. Knapp, on May 2, 1877, a girl, aged four years, previously healthy. First symptoms noticed about five weeks before examination, consisting chiefly in dullness, irritability, slight headache, and, on one occasion only, vomiting. Two weeks later internal strabismus (one eye) suddenly set in, and has persisted. No fever, spasm, or delirium. Previous to this attack there had been no emaciation, or cough, or ill-health of any kind. Dr. Knapp found double neuro-retinitis, with paresis of external rectus of one eye. On examination, I found the child with the above optic symptoms, and very cross; the buccal temperature was 37.2° C., and the pulse 96, perfectly regular. I made the diagnosis of non-tubercular localized basal meningitis, and expressed the opinion that the child's life was in no danger, though vision might remain considerably impaired. Dr. Knapp was giving potassium iodide, which I also advised. A few days ago Dr. Knapp informed me that a few weeks after I saw the child the strabismus disappeared, and that the neuro-retinitis gradually gave place to atrophy of the optic nerves, which, fortunately, was but slight, so that vision is now nearly perfect.

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* A Clinical Lecture delivered at the College of Physicians and Surgeons, New York, Saturday, February 23, 1878. From the *Hospital Gazette*, Mar. 1, 1878.

CASE III.—Sent me for examination by Prof. C. R. Agnew on February 14, 1878. I learned that the patient, a little girl five years old, had gone through an attack of chicken pox early in January, without fever or apparent ill-health. About January 19th the left eye "turned in," and strabismus has been constantly present since. No other symptoms have been observed—no fever, headache, irritability, etc. The mother states that one of her former children, at the age of eleven months, had convulsions and fever, became unconscious, and died in two weeks.

Examination of the eyes by Dr. Agnew reveals "double optic neuritis, with some stuffing of the disks; hypermetropia 4.5 of each disk."

I made the same diagnosis as in the second (first in point of time) case, viz.: local basal meningitis of non-tubercular nature. Advised blisters behind the ears, and large doses of potassium iodide.

These three cases illustrate a form of disease which is not, to my knowledge, treated of in the text-books, yet one which I suspect is not very rare, and which the more general use of the ophthalmoscope would render yet more common. Without a view of the fundus of the eyes in these little patients their trouble would have seemed strange or trivial. For, consider how few symptoms they presented.

Headache was present in two of them, and was frequent or severe only in one.

Vomiting occurred frequently in one child, only once in *Case II.*, and not at all in *Case III.*

Irritability and change in disposition (a real symptom in children, under many circumstances) occurred in only one child.

Strabismus occurred in all, and was, in reality, the only symptom which alarmed the parents, and caused them to seek advice. In *Case II.* Dr. Knapp expressly states that he found paresis of one external rectus, but in *Case I.* the muscles seem fairly strong, and it has occurred to me that perhaps, in these cases, the squint is due to a stronger accommodative effort which the child unconsciously makes to obtain better vision, as in the squint which accompanies hypermetropia. In *Case III.* there was hypermetropia of 4.5.

Neuro-retinitis was present in all the cases. Such important symptoms of intra-cranial disease as *convulsions, delirium, paralysis, fever, irregularity of the pupils*, and of the *pulse-rate* were absent in all the cases. In none of the cases was the basal disease secondary to any serious general fever or constitutional state.

The condition of the optic nerves and retina found in these cases is known as neuro-retinitis, or choked disks. In this state the optic nerves appear swollen, and may project considerably (measurably) above the level of the surrounding retina ; the margin of the disk is obscured or wholly lost, and no line of demarkation can be made out between the nerve and the retina. The blood-vessels present striking anomalies, the arteries being relatively small, the veins positively large and tortuous ; there are often small hemorrhages in the retina, round about the disk. This condition of choked disk may last a number of weeks (much longer in cases of tumor of the brain) and then subside, giving place to the appearances of atrophy of the optic nerves, viz.: an unnatural whiteness, or bluish whiteness of the disk, smallness of the retinal vessels, and unusual sharpness of the outline of the disk. A degree of atrophy must be looked upon as inevitable in the stage of recovery in cases such as those related above ; hence we must be cautious in prognosis *as regards vision.*

I would next invite your attention to the probable seat of lesion in these cases, and the mechanism by which choked disk is set up. At the base of the brain, anterior to the pons Varolii, and between the two temporal lobes, is a vast reservoir of subarachnoid fluid, contained in the meshes of the pia mater, in the so-called anterior subarachnoid space. Within this space lie the chiasm of the optic nerves, the roots of the olfactory bulbs, and the trunks of the third, fourth, and sixth nerves, on their way to the orbit. Each of these nerves, but more especially the optic, has a lymphatic circulation of its own, within its sheath, and in communication with the sub-arachnoid space. In reality, the same fluid which fills up the anterior sub-arachnoid space circulates in the lymphatic spaces of the optic nerves as far as the eyeball. When inflammation occurs at the base of the brain, or when a tumor is placed there, there is, of necessity, produced a retardation in the two circulations of the optic nerves—their blood circulation and their lymph circulation—and in consequence blood and lymph accumulate in the head (or retinal end) of the nerves, the arteries are small, the veins enlarged, and some of these may burst. Thus may all the optic phenomena of basal meningitis be explained. But, besides more active processes, exudation, and migration of leucocytes may take place in the delicate optic structures, and result in serious mischief. The effects upon the motor nerves are readily explicable by the same mechanical causation; but it is a little difficult to understand why the sixth nerve, which is certainly more robust than the fourth, should alone suffer. That is, upon the supposition that paralysis is actually present, and that we have not to deal with an accommodative squint.

We ought, with such a lesion so placed, to have some impairment in the function of smell. This is an interesting point which has not yet been investigated, I believe.

I have spoken of meningitis and tumor as giving rise to choked disks, and it may be well for me to say why I do not believe that tumors are present in these cases. *First*—Tumors of the basis are rare in children; they generally have intra-cerebral or cerebellar tumors of the tubercular kind. *Second*—A basal tumor will give rise to more positive paralytic symptoms than are present in our cases; either decided palsy of one or more cranial nerves, or weakness of the limbs on one side. *Third*—Convulsions would form a prominent feature in the symptom-group.

While regarding the lesion as an inflammation of the pia mater, I do not believe that it is tubercular, because of the absence of aggressiveness on the part of the disease, the absence of previous sickness, or of focus whence tuberculization might be set up; and, lastly, because in one case, recovery easily and rapidly occurred.

As to treatment, I would advise iodide of potassium in doses varying from .60–4. three times a day, well diluted. These little ones bear the iodide wonderfully well, when it is gradually increased. Counter-irritation has some effect at first, and I would place blisters behind the ears or on the temples—quite a series. At the same time I would give the child light but nutritious diet, keep it quiet, and avoid everything which produces determination of blood to the head, as active play, anger, surprises, etc. There is no need, I think, of confining the patient to the house.

Finally, gentlemen, I am pleased to speak to you of these cases, in order to give you faith in the value of the ophthalmoscope in the hands of the non-expert. I do not ask you to believe in the diagnosis, by any but our best oculists, of delicate lesions, such as slight anæmia or hyperæmia of the fundus of the eye, or faint atrophy of the optic nerves; but you, and all practitioners, should be able to recognize a fairly normal optic disk and retina, and to distinguish such gross lesions as choked disks, hemorrhage of the retina, and marked atrophy of the optic nerves. I trust that before you enter upon the practice of your profession, after finishing the elementary curriculum of the winter, you will seek private instruction in the use of the ophthalmoscope, and thus arm yourself with a weapon which will enable you to do more good, to improve your reputation by correct diagnoses, and, what is often more advantageous, to avoid damaging that reputation by an unfounded favorable prognosis in cases such as these, in which all signs, except the hidden ones, are not serious.

A CLINICAL AND THERAPEUTICAL CONTRIBUTION TO OUR KNOWLEDGE OF CERVICAL PARAPLEGIA.*

I PRESENT the following histories of cases to illustrate the semeiology of cervical paraplegia, and to demonstrate that the disease, in some of its forms, may be checked or even cured.

CASE I. Atrophic paralysis and anæsthesia in both hands; symptoms of weakness and stiffness in the legs; removal of these symptoms, and permanent arrest of the disease.

Dr. R——, U. S. Army, aged 41 years, first consulted me in 1873, and related the following history: Had always enjoyed good general health, never had syphilis, or been injured about head and spine, but had been much exposed to hardship and overwork in his army service. In December, 1870, he suddenly experienced diplopia with internal strabismus due to palsy of the left sixth nerve. This was not preceded or accompanied by neuralgia or by general symptoms. This affection gradually disappeared in the course of two months.

In July, 1871, the present disease made its appearance. Dr. R. was then in camp, and had been much exposed in severe marches. On rising one morning he noticed a marked degree of palsy in his left hand, the abduction of the thumb being impossible and opposition almost lost. At the same time both hands felt stiff and numb, though whether this was from palsy or from cold he could not decide. Dressing was very difficult that morning. The result of this attack was marked palsy of parts of the left hand and slight impairments of the motor functions in the right. The doctor cannot state positively what was the condition of sensibility in the right hand at that time. Three months later neuralgic pains appeared in the left thumb and forearm, and at the same time the thenar eminence on the left side began to waste rapidly. About the same time a diffused (corset-like) sense of constriction was noticed round about the chest, extending from beneath the clavicles to the lower ribs. At that time there was no palsy or wasting of the right hand; no symptoms in the face. The gait was unsteady, and fatigue easily produced. Closing eyes and attempting to walk in darkness made this unsteadiness greater. Late in the autumn of 1871 numbness (anæsthesia?) was first positively noticed in the ulnar distribution of the right hand, and this has since increased. At that time there was no numbness on the left side. This hand first became numb in the summer of 1872, one year after the palsy.

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* From the *Journal of Nervous and Mental Disease*, July, 1878, vol. v.

During the past six months the atrophy of the left hand has increased, as well as the anæsthesia. During this period the right thenar eminence has shown a beginning of atrophy about its centre (flexor brevis pollicis); the abductor indicis and the dorsal interossei also show some wasting. The fingers have been very awkward in use; the medius and annulus seeming to stick together. There has been much increase in the anæsthesia of the right hand. No general symptoms have appeared except moderate emaciation.

Examination.—The patient is a tall spare man of good muscular development, and no sign of disease except his wasted hands. Nothing abnormal about eyes, internally or externally. Facial muscles act well. On the left side there is a considerable area of partial anæsthesia in the range of distribution of the superficial branches of the supra-orbital, infra-orbital, and malar branches of the fifth nerve. Contact is hardly felt in this region, but painful impressions are perceived. The upper part of the trunk is the seat of much numbness, and some anæsthesia (?). At times it seems to patient as if a cuirass were round about him, extending as low down as the false ribs and the umbilicus. No such sensation lower down.

Both hands are the seat of muscular atrophy and anæsthesia distributed as follows: The left hand is extremely wasted. The small muscles of the thumb have disappeared, with the exception of the inner part of the flexor brevis and adductor. Complete extension and opposition of the thumb are impossible. The muscles of the hypothenar eminence and the interossei are uniformly wasted. The atrophied muscles yield no contraction to the strongest faradic current, and only slight fibrillary contractions to the interrupted current of thirty-two elements of Stöhrer's galvanic battery.

The right hand exhibits a very moderate wasting of the interossei, and a narrow streak (6 mm.) of positive atrophy in the abductor and opponens pollicis. The interossei controlling the index and medius fingers are most affected. The various muscles of the hand, excepting the atrophied band in the thenar group, respond well to both currents. The handwriting is much altered and laborious, the patient feeling as if the difficulty were one of incoordination, though this is not strictly correct. On neither side is there the main-en-griffe which is so characteristic of extreme palsy of the interossei. The forearms and the rest of the body are free from paralysis or atrophy.

Sensibility is much impaired in both hands, but more in the right; so that we see in this case an imperfect example of the phenomena accompanying a lesion in one half of the cord. The degree of tactile anæsthesia is great, but pain and temperature are everywhere perceived when the stimulus is sufficiently strong. In the right upper extremity the loss of sensibility is in the inner (ulnar) side of the lower arm, forearm and two-thirds of the hand, the whole of the fingers and part of the thumb anteriorly. The left upper extremity exhibits a patch of anæsthesia a little different in shape. Anteriorly, the inner (ulnar) half of the lower arm and of the forearm is slightly anæsthetic, and the same is true of the same parts of the posterior aspect of the forearm and hand. In the hand the anæsthesia almost follows the distribution of the ulnar nerve.

There is a faint feeling of numbness in the remainder of the upper extremities as high as the acromion processes, but there is no true anæsthesia.

In the lower extremities there is nothing objectively abnormal. At times there is marked uncertainty in walking, patient feeling in danger of staggering against persons and things. There is no ataxia, and while standing with eyes closed no great oscillation. The doctor was formerly a great walker, but now he is easily fatigued by half a mile of promenade. No rectal or vesical symptoms.

Diagnosis.—I reject progressive muscular atrophy, because of (1) the paralytic onset; (2) the occurrence of anæsthesia; (3) the limitation of disease to the hands; (4) the want of symmetry in the wasting. There was probably a small hemorrhage in the spinal cord at the time of sudden paralysis of the left hand. If there was a clot it must have been very small, and was located in the left anterior gray horn in the middle of the cervical enlargement. From this focus a myelitis has extended in a direction chiefly downward and across the median line. The most remarkable feature of the case is the grouping of symptoms in the order assigned by Brown-Séquard to spinal hemiplegia, *i.e.*, more palsy on one side (same side as lesion of spinal cord), and more anæsthesia on the opposite side.

During the autumn and early winter of 1873 I treated Dr. R. systematically. The local treatment, having for its object the improvement of the atrophied muscles, consisted in thorough galvanization of the parts, friction, etc. A few fibres of the left thenar eminence seemed to revive and grow after weeks of patient care, but no real progress was made.

It was otherwise with the internal treatment. Under the use of nitrate of silver, arsenic, cod liver oil, etc., the myelitis was undoubtedly arrested. Before leaving for the Pacific coast early in 1874 Dr. R. could walk much, felt less inclination to stagger, was much less conscious of the cuirass feeling, and gained a great deal in general vigor.

Dec. 1, 1875. In the last eighteen months no medicines have been taken, yet the disease has made no progress. The hands are about the same, he has hardly any sense of constriction about the thorax, he walks perfectly well, and his health is good.

Looking over the history and progress of the case in the light of recent discoveries in the pathology of spinal paralysis, I am disposed to modify my first diagnosis somewhat. I adhere to my denial that the case was one of progressive muscular atrophy, but doubt if at any time there was hemorrhage in the cord. The original lesion may have been a rapidly developed limited myelitis in the left anterior gray horn in the lower cervical region, with subsequent chronic myelitis in various directions, chiefly across the median line, backward and downward. The case bears a certain resemblance to cases of acute spinal paralysis in the adult. The weight of evidence is very nearly equally in favor of both my hypotheses.

1877. I have several times met Dr. R. in the last two years, and he has always expressed himself as perfectly well except in his hands, which remain as they were in 1873. All signs of active myelitis have long since disappeared, and we may assume that the disease has definitely come to a stand-still.

Case II.—Atrophic paralysis in both hands with slight anæsthesia; neuralgic pains in both arms; weakness of legs. Treatment by active counter-irritation, mercury and iodide of potassium, galvanism; cure.

Mrs. H., aged 53 years, was brought to me by my friend Dr. Conrad, of this city, on July 30, 1877. I obtained the following history: Some time during January of the present year she began to experience pain and numbish sensations in the tips of the index and medius fingers, later in thumb and palm of hand. There were no abnormal sensations in the ring and little fingers. The left hand alone was at first affected. She thinks that previous to January she had had some pains in arms, but cannot describe them. These pains (those occurring in late winter) were followed by weakness and wasting of the hands.

In May, when seen by Dr. Conrad, there was the following condition: The right hand was only a little weak; the left was the seat of neuralgia and numbness as above described, the left thenar muscles were atrophied, and there was marked loss of power. Since that time the pains have been more clearly neuralgic, following the course of the median and ulnar nerves from the palms to the bends of the elbows, occurring in paroxysms every ten or thirty minutes, very seldom affecting both arms at the same time. There seemed to be more pain on the right side. About the middle of June numbness showed itself in the tips of the medius, index, thumb and palm of right hand. No numbness in range of radial and ulnar nerves. Closing hands has made the pain worse; feeling in fingers and palms is a sore, scalded sensation. Has had some pain in the middle of the back below the shoulder; no spinal pain strictly speaking; no cerebral symptoms; no numbness in feet, but legs *have been weak;* no spinal epilepsy or cincture feeling. Complains of slight dysphagia.

Dr. Conrad has given the patient small doses of biniodide of mercury and iodide of potassium, and had applied an ascending stabile galvanic current from the hands to the back of the neck.

Examination.—Slight but distinct tactile anæsthesia in fingers supplied by median nerves (including inner half of annulus); for example on the tip of the medius finger the points of the æsthesiometer are distinguished only at 4–5 mm. apart. There is marked paresis of both forearms and hands. Left hand tremulous. The only atrophy visible is in the outer part of the left thenar eminence. The other muscles are weak but not visibly wasted. A faradic current applied to the median and ulnar nerves (nerve current) produces contractions in all muscles except the part of the left thenar eminence which is wasted. The median nerves are not tender or unduly irritable under pressure in any part of their course.

The patient has had several miscarriages and other symptoms which justify a suspicion of syphilis. Has been taking 2. of iodide of potassium a day, and galvanism.

I made the diagnosis of central myelitis in the upper part of the cervical enlargement, chiefly in the left half of the organ, involving the anterior gray horn. My advice was to insure absolute rest for the hands and arms, patient not even to feed or dress herself; to apply mercury by inunction, and to give the iodide of potassium in much larger doses. The actual platinum cautery was shortly applied over the upper cervical vertebræ.

August 11. Again seen with Dr. Conrad. Patient is no worse as regards pain and atrophy. The legs are perhaps weaker. Mercurial ointment to the amount of 40. has been used without effect on the gums; has had 9. of iodide a day. Advise repetition of cautery every other day, the use of 12. iodide per diem, more inunction, also 4.cc. of Squibb's fluid extract of ergot at bed-time. Mrs. H. has been rather careless in respect to resting hands. She is strongly urged to do nothing whatever with them. Very mild galvanism to arms and spine.

Sept. 11. Patient is better in some respects. There is no increase of wasting, less pain and dysæsthesia in fingers. Legs are weak, but without increased reflex. In the night the hands become clenched. A little tremor is observed in the lips. Treatment has been faithfully pursued; counter-irritation, rest, biniodide of mercury and iodide of potassium in large doses; ergot up to 8. at night. The gums have been kept a little tender. During the winter iodide of potassium internally, and galvanism to the hands and spine in the shape of the ascending stabile current, constituted the treatment. Almost absolute rest was enforced. Improvement appeared and continued.

March 9. Seen with Dr. Conrad. Patient is practically cured. The muscular masses of the hands are fully restored; grasp is good. Very little neuralgic pain is now felt in the arms, but lately some pain has appeared in elbows and knuckles. There is a mere trace of numbish feeling in the fingers. Legs only feel weak after going up stairs. Continues iodide and galvanism.

A few weeks ago, in May, I met Dr. Conrad, and he informed me that with the exception of occasional neural pains, his patient was perfectly well.

In concluding this case I would express my thanks to Dr. Conrad for his courtesy in allowing me to make use of it, and would congratulate him upon the skill with which he carried out the treatment agreed upon.

CASE III.—Extreme anæsthesia of both upper extremities and of the upper part of the trunk; atrophic paralysis of the right hand and of many muscular groups of the left upper extremity; contraction of the left pupil. No symptoms in the lower limbs except rigidity in the left leg. Central myelitis in the cervical enlargement of the spinal cord, with probable formation of a cavity.

Annie M——, single, aged 23 years. Seen at the Manhattan Hospital, May 18, 1878. When twelve years of age patient's ankles were weak for two years, but entirely recovered. Five years ago, when eighteen years old, she noticed numbness in the tips of the fingers of both hands, extending to the shoulders in the course of a few months. The legs were not numb. Next there began wasting of the left shoulder, hand and arm. Since at least two years the left arm has hung useless by her side. The right hand has wasted more recently. Has felt fibrillary contractions from the start, and believes that sensibility was early lost in the hands. The left leg is weak, and in the last three months it has been getting very stiff. At night her whole body jerks. Micturition is only slow ; constipation is present ; menses are regular. No dyspnœa or palpitation. Has some occipital headache. According to patient's statement, feeling in the feet and legs is normal, though at times the left foot tingles. Has never had neuralgic pains in arms or legs. General health good.

Examination.—Both pupils are small and equal in a bright light ; in the shade the left does not expand, while the right does. No paralysis, atrophy, or anæsthesia in the face. Face not flushed. The upper part of the thorax and the shoulders present marked anæsthesia and analgesia ; in the upper part of the back and shoulders behind, sensibility to touch and pricking is fair. In the arms, forearms and hands sensibility is wholly lost ; patient has often burned herself without knowing it. The left upper extremity is extensively paralyzed, while the right is only partially so in the hand. State of muscles : On the right side only the interossei are weak and wasted. On the left side the following muscles are atrophied and palsied : interossei, biceps, brachialis anticus, supinator longus, supra and infra spinati, and deltoid. The flexors and extensors of fingers and wrists, the triceps and pectoralis are simply feeble. The scapulæ are not winged ; there is a slight dorsal spinal curvature, convexity to the right ; no kyphosis ; standing with eyes closed is difficult ; left lower extremity is the seat of increased reflex and epileptoid trepidation. No atrophy or paralysis of lower limbs. Measurements : right calf, 32 c.; left, 31 c. Tendon (knee) reflex increased on both sides.

Re-examined in bed, May 19, 1878. The face presents only the symptoms above noted. The upper extremities, as high as the insertion of both deltoids, are perfectly anæsthetic to touch, reasonable pricking and pinching, and to firm grasp. On the upper thorax and back on the shoulders, she feels touch fairly well, but pricking very little. In front, normal sensibility reappears at the level of the fourth rib. On the back the limiting line is indistinct and seems to be somewhat below the spines of the scapulæ. Motor symptoms in arms as above. The lower limbs and the abdomen present no anæsthesia. Legs and thighs are well nourished ; left calf very firm ; left toes are "en griffe"; foot not inverted. Patient's mother states that her left leg is very stiff in bed and on first rising in the morning, but after walking it becomes more limber. Patient denies having a cincture feeling or dyspnœa. When numbness appears in the left leg (rarely) it extends to the knee, but patient qualifies the statement by saying that the feeling is more like cramp. At the age of twelve years it would seem that patient had an attack of palsy in the left leg below the knee ; the leg and foot were swung heavily for awhile. At that time the left arm was not affected. She recovered perfectly in two years. Every symptom now observable in the left leg and foot has appeared within the past year. The temperature of the hands, taken for three minutes with a Casella thermometer, placed between the index and medius, is, on the right side 34.7° C, on the left 35.° C.

Electrical examination, June 3d. Faradic current, right upper extremity. All muscles give good reaction except the outer group of dorsal interossei. Left upper extremity, good reaction in trapezius, pectorals, triceps ; proper extensors of wrist and fingers ; faint reaction in long flexors of wrist and fingers, inner third of deltoid, opponens pollicis ; no reaction in hypothenar group, inner part of thenar group, interossei, supinator longus, and biceps.

It is plain that in this case there is a great lesion in the cervical enlargement of the spinal cord; probably a diffused central myelitis with formation of a cavity. The lesion was first developed in the æsthesodic tract of the cord, and is yet greater there than in the kinesodic system, though the anterior horns, especially on the left side, have become involved. The cilio-spinal centre in the left side of the cord is injured, as shown by the contracted pupil. It is noteworthy that no symptoms of vasomotor paralysis are present, thus affording a demonstration of the independence of the cilio-spinal and facial vasomotor centres. The symptoms in the left lower extremity point to the existence of secondary descending degeneration in the lateral column.

It is very remarkable that with so much disease in the cervical enlargement, the various nervous conductors for the lower limbs and abdomen should not be interfered with. As regards the uppermost limit of the lesion, that cannot be above the origin of the fifth cervical nerve, as the diaphragm acts perfectly.

A few words as regards the pathology of these cases.

In all three the æsthesodic, kinesodic, and musculo-trophic tracts in the cervical enlargement were affected.

In Cases II. and III. the affection was probably inflammatory —perhaps syphilitic in Case II. In Case I. a doubt may exist as to whether hemorrhage took place, or whether there was a suddenly developed (as in infantile poliomyelitis anterior) inflammatory lesion. Even if there was hemorrhage at first, a secondary adjacent inflammation occurred and presented many of the symptoms. I am disposed to believe that a central cavity has formed in the cervical region in Case III., because of the resemblance of this case with the cases of central myelitis with formation of cavities reported by Schueppel, Hallopeau, and others.

As regards therapeutics, the exceedingly satisfactory issue in Case II. was perhaps obtained because the lesion was essentially syphilitic, and the proper remedies were freely and persistently used. Yet I am disposed to attribute much of the recovery to the almost absolute rest enforced. On careful consideration, in view of the apparently progressive nature of the lesion, the issue in Case I. is almost as gratifying. True, the atrophied and anæsthetic hands were not restored, but symptoms which seemed to point to approaching general paralysis were permanently dispelled. Case III. has been but a few days under treatment, and is a very unfavorable one.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF DISSEMINATED CEREBRO-SPINAL SCLEROSIS.*

MR. PRESIDENT AND GENTLEMEN:—This thesis is based upon two cases. Case I. came under the observation of Dr. Van Derveer, of Albany; the history is as follows:

Thos. Grogan, accountant, æt. 29, unmarried. Mother died at the age of 49, during her climacteric period, from the exhaustion following sciatica. One brother died of phthisis pulmonalis at the age of 26. Several brothers and sisters died during infancy. The father, a brother and a sister are still living, and in good health. Saw the case first in Feb. 1870, when the following facts were elicited:

Habits have always been good; no sexual excess or masturbation, and has not been exposed to venereal diseases. Has been quite studious and had acquired a good education. Was fond of society, and spent many of his evenings at dances up to the time he was taken sick.

In the latter part of December, 1867, his attention was first attracted to a sharp pain about an inch back of the right eye. He was impressed with the idea that he had strained the eye and began wearing a pair of spectacles. While attempting to hang a picture, he noticed that he could not see it distinctly enough to determine whether it was hung properly or not. In April, 1868, had frequent nocturnal emissions, but did not apply for treatment. While attending a military funeral in June, of this year, with a prominent organization of this city, he experienced no trouble in his power of locomotion until he had walked about five blocks, when he began to stagger like a drunken man, and after several ineffectual attempts to march steadily, was obliged to leave the ranks. He immediately returned home, and consulted his family physician, who thought that he was probably suffering from a partial sun-stroke. No particular treatment was ordered aside from rest, and partially recovering his strength he returned again to his office duties. In August he was much annoyed by a feeling of internal warmth in the lower part of the left leg, but adopted no treatment. Went to Pennsylvania in October to secure rest and recruit his health. Any unusual excitement at this time would cause severe nervous tremors and a sensation "akin to creeping of the flesh." During his visit he suffered much pain in the left ankle and thought he had sprained it, but could not tell when or how he had done so.

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* By Dr. E. C. Seguin, New York, in collaboration with Dr. J. C. Shaw, Brooklyn, and Dr. A. Van Derveer, Albany. (Reprinted from Journal of Nervous and Mental Disease, April, 1878.)

He returned to his home about November 1st, and applied himself closely to business until the middle of February, 1869, when his strength failing he gave up business by the advice of his physician. Was confined to the house during three weeks, regarding his weakness as due to overwork. During the winter of 1868–'69, Dr. Alden March applied the moxa several times to his spine. At the close of his three weeks' confinement he was again able to take exercise in the open air, and walked with the aid of a cane. In the course of four or five weeks he was again much prostrated, so much so that he himself entertained serious apprehensions concerning his recovery. He was now troubled with more or less flatulence, dizziness, loss of sight and constipation. Improved somewhat in May, and continued so until August. Went to Sharon Springs and took sulphur baths up to 37.75° Cent. daily during four weeks, and drank at the same time from one to two quarts of sulphur water each day. This produced great prostration, and he was brought home in four weeks greatly debilitated, and much reduced in strength and flesh, and now for the first time in the history of his case was he unable to walk without support. He first came under my observation in February, 1870, when he presented the following more prominent symptoms: Strength somewhat improved since his return from the springs, and he can now, with great effort, walk alone, but gravitates in an irregular way from side to side while doing so. Complains of great weakness in his arms and legs, and a feeling of great constriction about the body. Cannot see to read, but can distinguish a friend at a distance across the street. His feet are very sensitive to tickling. Standing with his feet together, when his eyes are closed, he thinks he would fall if left alone. Will not trust himself to come down stairs without watching his feet closely, and feels a sensation as if walking upon cushions. Drags his feet when walking. Can converse with little or no effort. Enjoys conversation, is humorous, and fond of quoting from Shakespeare. Is unable to whistle on account of a spasmodic twitching of the muscles on the right side of the face when he attempts it. Can put out his tongue in all directions. Very difficult for him to attempt to pronounce the words, "truly moral." Generally sleeps well the latter part of the night, bowels constipated; pulse and respiration normal. Urine is passed without trouble, and at regular intervals in usual quantity; is acid, has a specific gravity of 1020; is heavily loaded with phosphates. Has seminal emissions from four to six times a month. Surface of skin is very hyperæsthesic. Upon strong percussion along the spinal tract, feels some pain. Ordered 1.2 cc. fluid extract ergot three times daily, with good, generous diet, also directed him to take an occasional saline cathartic. Continued this treatment for three months and he thought he was improving, but, in fact, had not so good use of his limbs as before.

June 1. Can whistle somewhat more distinctly, but complains that he is losing control of his arms. Feeds himself with difficulty. When asked to touch his nose with either index finger, his hand describes a semicircle, and as he approximates the finger to the nose, it is done with a sudden jerk.

Ordered a pill, consisting of .002 strychnia, .015 capsicum, .06 reduced iron and, as he does not sleep, 1. bromide of potassium, to be taken at bed-time. Has frequent erections and great sexual desire. Urine still abundantly phosphatic in character. Continued the above treatment until Sept. 1st, with no amelioration, and a gradual loss of power of co-ordinating the lower limbs. Unable to walk alone, and walks with difficulty when supported; drags his legs along, advancing the heel first with a jerk. Ordered phosphor. acid and strychnia, with bromide of potassium at night.

Dr. Clymer saw him about October 1st, 1871, and observed the following more marked symptoms in his disease: Tone of voice drawling; brain seems weakened, and nystagmus of the eyeballs is apparent. Has, in addition, spinal epilepsy; it having only a general connection with the sclerosis. The latter condition only occasionally present. Sensation in both limbs impaired. The muscular will-power in the right limb is good, but much weakened in the left, and in the latter, sensation is confused and tardy. Sensation of heat or cold, or the impression of a sharp instrument, reaches the brain much sooner from the one part than another. There is characteristic dragging of the feet, and the will-power is inadequate to give the proper stimulus to the muscles, yet he displays considerable control of the leg when attempting to extend or flex it. Took most of the time until October, 1872, a pill consisting of .0012 phosphorus, .002 strychnia, .06 reduced iron, but with no apparent benefit. October 1, 1872, Dr. Clymer saw him a second time. The spinal epilepsy still continues, but is not so marked. The muscular will-power is much weakened. Has lost much in flesh, and at times there have been well marked symptoms of paralysis of the right side of the face. Cannot whistle and talks very indistinctly at times. Urine phosphatic in character and bowels much constipated. No nocturnal emissions; no sexual desire. Anæsthesia is very decided and sensation confused. Little treatment resorted to from this time until his death, except to keep his bowels regulated, and occasionally quinine to improve his appetite. Has to be fed, as he cannot carry anything to his mouth, having little if any control of his hands. Sight much impaired. Three months before his death his urine was withdrawn several times, and then again a week previous to his death. It became very difficult for him to talk three months before his death, but at no time were well marked symptoms of aphasia observed. His trouble in speaking seemed to be due to paralysis of the muscles. In Sept. 1871, Dr. Stevens examined his eyes with the ophthalmoscope and diagnosticated sclerosis of the optic nerve.

Patient died Feb. 21st, 1874. At the autopsy—no general post-mortem being allowed—only the brain and spinal cord were examined, which were removed entire and sent to Dr. Seguin immediately.*

Case II. came under Dr. Seguin's observation.

Female, æt. 23. Single. Seen Oct. 20, 1873. A nervous girl, with occasional irregularity of menstruation, but no dysmenorrhœa. At times hysterical laughter and tears; never convulsive attack. In July, 1871, while out walking, after having climbed a number of walls, felt weak and awkward in right leg; thought she had sprained her knee. There is not enough evidence to support this statement. Ever since she has had weak right leg, without anæsthesia or numbness ; at times more use of leg than at others ; almost cured once or twice ; of late has required help of crutch, or friend's arm in walking. When I examined Miss P., I found paresis of right leg, the loss of power being marked at ankle and toes. There was doubtful weakness of the right hand. I could not make out that the knee joint was affected. The muscles of the right leg showed a slight diminution of reaction to the faradic current, and this agent also showed that sensibility to pain was a little dull in leg and foot.

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* History by Dr. Van Derveer.

In view of the history of the case, the capricious development of the palsy, the absence of reliable signs of central disease, the presence of a strong neurotic element in the family, and the fact that strong emotions had been acting upon her, I concluded that the patient had a functional palsy of an hysterical nature. Strychnia was given her and faradism used. The specific effects of strychnia appeared and the patient was decidedly tetanized for a while ; this passed off, and when I last saw the patient, on December 11th, she was in about the same state as at the beginning of treatment. The unfavorable effects of the treatment led me then to believe that the patient had an obscure central lesion, probably sclerosis. In March or April, 1874, patient rapidly grew worse, becoming paraplegic, and her hands showing paresis. In July she was placed in an irregular water-cure house, where extensive bed-sores formed in consequence of want of care and of cold applications to the palsied parts. (She had continuous applications for several days.) Exhaustion and pyemia caused death, August 1st. The post-mortem examination showed disseminated sclerosis of the spinal cord. The brain not examined. Dr. Charles A. Leale, of this city, treated the patient during July, after the bed sores had formed, and I made the autopsy at his request and that of Deputy Coroner Dr. Shine.

After hardening in bichromate of potash, sections were made in various regions of the encephalon and cord in Case I. In the brain proper, small patches or nodules of sclerosis 1 to 3 mm. in diameter exist in various parts of the white substance of the hemispheres ; and there are a few just under the gray cortex. In the right nucleus caudatus, near the posterior margin, are several nodules ; in the right occipital lobe just outside of the posterior horn of the lateral ventricle, extending well back towards the convolutions at the apex of the lobe, is a long sclerosed patch.

Pons and Medulla.—The upper part of the pons Varolii and crura have not been critically examined. In the medulla oblongata the sclerosis appears in the following parts :

1. At the level of the apex of the fourth ventricle (Fig. 5), and below, a patch about 2 mm. exists in the very centre of the section across the median raphe.

2. In a section made at a point 6 mm. above the apex of the fourth ventricle (Fig. 6), a large patch of sclerosis invades the floor of the ventricle, including the mass of gray matter which gives origin to the hypoglossus, par vagum, and glossopharyngeal.

Few cells of the hypoglossus nucleus are visible, and these are small and rounded.

3. In a section made through the point of origin of the 6th and 7th nerves at about 2 mm. above the inferior border of the pons Varolii, the sclerosis is found in about the same location, viz.: round about the nucleus of origin of these nerves under the floor of the fourth ventricle (Fig. 7).

Cord.—Sections have been made in the cervical, dorsal and lumbar regions, stained in carmine and hematosin, and mounted in Canada balsam. With a low power or with the naked eye the following distribution of the lesion can be made out:

In the cervical region (Fig. 1), the sclerosis involves the following districts: almost all the right anterior column, and the entire anterior horn, the posterior part of the left anterior column, and the whole of the anterior horn, the posterior part of the lateral column, a narrow band of cortical sclerosis of both posterior columns, the lower part of the columns of Türck near the commissure; all the gray commissure involved in the disease.

In a section a little below this in the *cervical region* (Fig. 2) the location of the lesion is somewhat different. The most decided disease is confined to the anterior part of the left anterior column, a small part of the external edge of the anterior horn, the posterior part of the lateral column, the columns of Goll and the gray commissure.

In the dorsal region (Fig. 3), decided sclerosis of outer part of left anterior column and the whole of the anterior horn, the columns of Türck, and a slighter sclerosis of all the cord posterior to a line drawn through the central canal.

In the lumbar region (Fig. 4), the right anterior column and border of the anterior horn, the posterior part of the right lateral column, both posterior columns, gray commissure, and slight sclerosis of the periphery of left anterior horn.

In Case II., only the spinal cord was obtained.

In the cervical region (Fig. 8), the following districts are diseased: the columns of Türck, posterior part of right anterior

Fig 1.

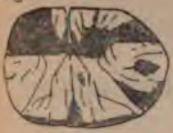


Fig 10.

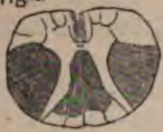


Fig. 11.

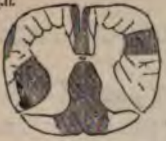


Fig 12

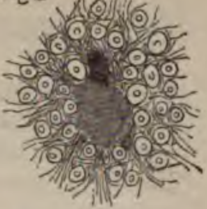


Fig 2.

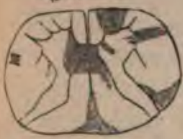


Fig 15.

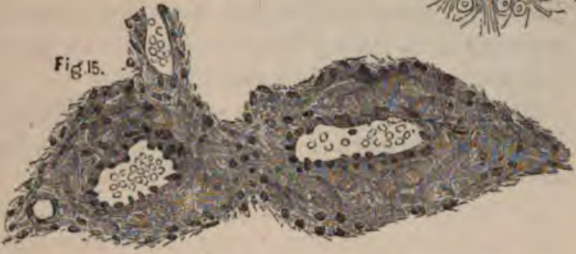


Fig. 3.

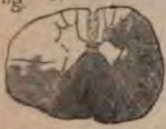


Fig. 4.

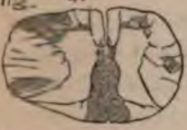


Fig. 13

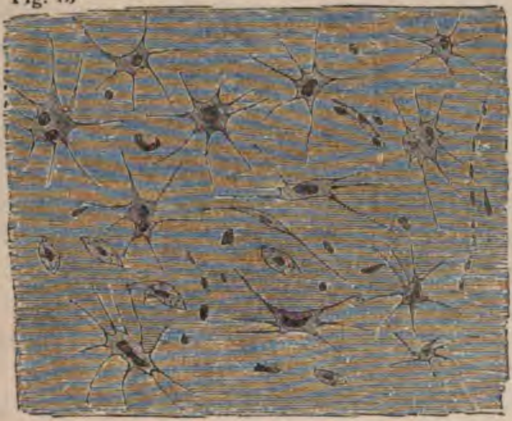


Fig. 5.

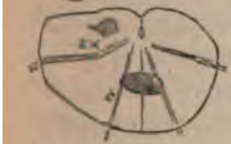


Fig. 6.

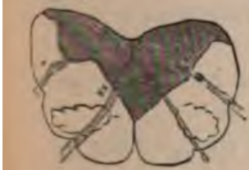


Fig. 7.

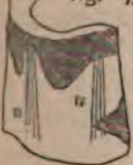


Fig. 14

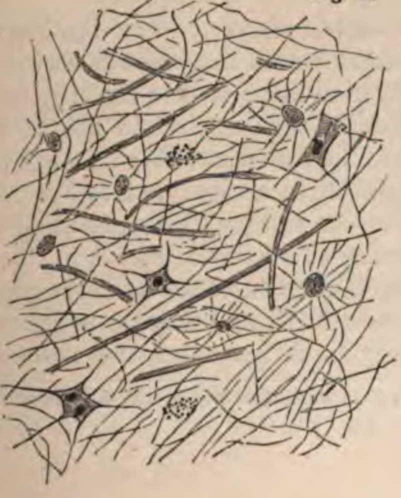


Fig. 16

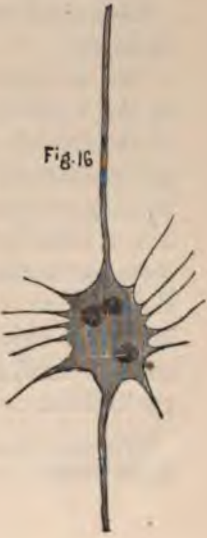


Fig. 8.

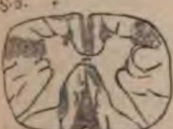
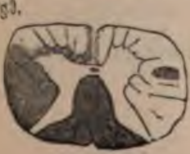


Fig. 9.



column, the upper part of right column of Burdach, the columns of Goll, posterior part of left anterior column.

Sections just below above in the cervical region (Fig. 9) show the following distribution: the columns of Türck, part of right anterior and lateral column, the entire posterior columns, the periphery of the right anterior horn.

Dorsal region (Fig. 10), columns of Türck, the periphery of both anterior horns; the gray commissure, both lateral columns and both posterior columns.

In the lumbar region (Fig. 11), the columns of Türck, right lateral column, posterior part of left anterior column, entire posterior column, the periphery of both anterior horns. It will be seen from this description that the sclerosed nodules are of various sizes, and occupy the most diverse regions of the cerebro-spinal system, involving the gray as well as the white matter.

Histology.—The histologic study will include the two cases.

For convenience of study and description we will divide the morbid process into three (3) stages. This division, although arbitrary, can nevertheless be observed on a close study of the specimens, and aids very much in a description and the understanding of the various conditions which are observed in the many diseased regions of the cerebro-spinal substance:[*]

The first stage will comprise the very earliest changes which are to be observed.

The second stage, in which the morbid process has advanced considerably.

The third stage, in which the most extensive changes are to be seen.

The Neuroglia.—First stage. An examination of the neuroglia of the white matter at this stage shows an increase in the size of the nuclei; their number is also slightly increased; there is an increase of the protoplasm around the nuclei, the quantity varying very much in different cells; in most of the cells it is only a little greater than normal, in a few it is enormously increased, as can be seen in Fig. 12, representing two cells seen in the anterior columns of the dorsal region in Case I. The nuclei have not only undergone increase in their size, but they have also assumed the most diverse shapes; in those cells where the protoplasm is very much increased, the nucleus is found at the extreme edge, as if the protoplasm had increased all on one side; we also begin to notice slight processes from the protoplasm; even now there is an appearance of increase in the size of the fasciculi of tissue running between the various areas of nerve tubes. At this time all the surrounding nerve tubes appear to be normal, their axis cylinders and myelinic sheath are perfectly distinct. In the gray matter at this period, the alterations are very similar: a slight increase in the size of the nuclei and the protoplasm around them, and beginning to be visible processes from the protoplasm.

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[*] The medulla and pons were examined by Dr. Seguin, the two cords and brain by Dr. Shaw.

In the second stage, the nuclei and their surrounding protoplasm have increased in size, their processes are now much larger, and more distinct and apparently more numerous; and there is altogether a much larger number of these branching cells visible. There is now present a diffuse granular appearance; no special fibrillation to be seen; a very close observation shows that the axis cylinders are present, but that their myeline sheaths have gone. (The granular appearance at this period is very probably due to the breaking up of the myelinic sheath into a granular-looking material which becomes diffused among the surrounding tissue, and this perhaps also explains why we do not see the increased fibrillation of the neuroglia.) The process at this period is the same throughout the brain and cord; in the gray as well as in the white matter.

In the third stage these branched cells have grown to an enormous size, their processes are numerous and sometimes of immense thickness and length (Fig. 13); now very little granular material is present, and we see distinctly the enormous increase of neuroglia fibres, and which are of exceeding coarseness; there appears to be no evidence of anastomosis between them. In a stage a little advanced of this (Fig. 14), these branched cells appear much less numerous, they are less distinct, their nuclei are less sharply stained, their outlines are not so perfect, and they sometimes appear as imperfect bodies; they in their turn appear to have lived their day, and are now undergoing decay.

Nerve Fibres.—In the second of the artificial periods which we have created for convenience, we see the first changes in the nerve fibres; their myeline sheath is undergoing disorganization; but their axis cylinders still remain.

In the third period, when the increase of neuroglia fibres is at its greatest height, we still see among its coarse fibres numerous axis cylinders, in good preservation (Fig. 14). This preservation of the axis cylinders is considered almost distinctive of disseminated sclerosis by Professor Charcot. Charcot states that the axis cylinders have become hypertrophied; this is a matter which appears open to question.

Ganglion Cells.—The following description holds good for the ganglion cells in the entire cord. In the decidedly diseased portions there is a remarkable absence of processes; but it happens occasionally, even in a markedly sclerosed area, that a cell will be seen whose process is apparently normal, and can be traced for a considerable distance; most of the cells contain more or less yellow pigment, some are almost entirely filled with it, and no nucleus is visible; in other cells the pigment is seen encroaching on the nucleus; in some cells the nucleus is seen with clear and definite outline; in many other cells they present an indistinct jagged edge, and occasionally they appear to be making an effort at budding.

In some cells all the nucleus appears to have undergone a form of dissolution, as it presents a very broken appearance. In a number of cells the nucleus is displaced, sometimes almost into one of the large processes, in others to the extreme edge of the cell. In a markedly sclerosed area in the anterior horn of the cervical region, the cells have undergone a simple diminution in size, so that they are reduced to at least one-third. The cells in the columns of Clarke have undergone the same changes. The alteration in the cells is greatest in Case II.

Vessels.—In Case I. the muscular coats of the arteries are very much hypertrophied, there is also some increase in the intima and adventitia and an increase in the number of nuclei; the lumen of the artery is therefore rendered much smaller than normal. (Fig. 15.)

In Case II. the arteries have also undergone the same changes; although the muscular coat is not nearly as much hypertrophied, and the lumen of the artery is much larger.

In comparing the arterial changes in these two cases we find that in Case I. the arterial change is greatest, and the sclerosis less marked, whilst in Case II. the sclerosis is most decided and the arterial change much less.

The nuclei of these branching cells are very sharply stained by the hematosin, whilst the body is very lightly stained; they have slightly granular contents; these nuclei present the same appearance at all stages of the process. The cells contain one, two, three and even four nuclei of irregular shape; and often nuclei are seen with partial constrictions on them as if they were about to divide; the nucleus is most usually placed to one side of the cell. The processes which are given off from these cells are very numerous, and always leave the cell by a broad base, tapering off to a hair-like extremity, which becomes lost in the surrounding tissue. The shape of the cells, and the manner in which the processes are given off, varies very much; some cells are to be seen with only two processes, one at each end.

Other cells have a rather long body, and many processes given off from each end; but the majority of cells have processes from all sides. Two of the cells seen are of enormous size, having a process of very great thickness, which can be seen for quite a distance and does not taper off like the other processes. (Fig. 16.)

There are besides these branching cells small nuclei in more or less large number and of a variety of shapes; but mostly round and sharply stained. These curious branching cells have been seen by other observers. Lubimoff[*] and Mierzejewski[†] have described them in the cerebral substance of general paralytics, Charcot and Gombault in a case of syphilitic disease of the protuberance, Pierret in a case of myelitis. Debove appears to have seen them in a sclerosed patch of the ependyma ventriculorum. They are to be seen in a case of central myelitis with cavities from a patient of mine.

They have been figured by Jastrowitz[‡] in an article on "Study of Encephalitis and Myelitis in young children." Adler[§] has also figured them. The cells figured by Jastrowitz are very similar to those seen by us. Mierzejewski describes these cells as being connected with the walls of blood-vessels. We have observed them in the vicinity of blood-vessels and sending their processes towards them, but it is very difficult to determine in this case whether they unite with the wall of the vessel or not. They have no special predilection for the neighborhood of vessels, but are to be seen everywhere. Pierret describes them as anastomosing; we have not seen any anastomoses between our cells.

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[*] Lubimoff, Beitraege zur patholog. Anatomie der allgem. progressiven Paralyse, *Archiv für Psych. und Nervenk.*, Band 4, p. 579, 1874.

[†] Mierzejewski, Études sur les lésions cérébrales dans la Paralysie Générale, *Archives de Physiologie*, Tome 2, 1875; 2d Series, p. 195.

[‡] Jastrowitz, *Archiv für Psych. und Nervenkrankheiten*, Band 3, 1872, p. 162.

[§] Adler, Ueber einige path. Veraenderungen an den Hirngefaessen Geisteskranken, *Archiv für Psych. und Nervenkrankheiten*, Band 5, 1875, p. 77.

Frommann figures, in sclerosis of the cord, cells somewhat similar; but they have fewer processes and are very much smaller than the cells observed in our cases; 500 to 950 diameters made them distinct; their protoplasm is not so abundant.

The interest attaching to these cells is in knowing if they are normal elements of the neuroglia which have become hypertrophied. In looking over the records of pathological cases which have preceded, and the many recent observations in normal histology of the neuroglia, there appears good reason for believing that these are really normal elements of the neuroglia which have become hypertrophied. The cells, with hair-like processes, figured by Boll* and Deiters. Those depicted by Frommann, although containing a much larger number of fine processes and a nucleus surrounded with very little protoplasm, might be supposed to have undergone a modification so as to present the appearances of our cells.

Butzke† figures cells with processes from the normal neuroglia, which, on the whole, have a much closer resemblance to the cells just described by us than any others we have seen. His cells have a good-sized nucleus with a considerable amount of protoplasm around them, and numerous fine processes with one or two large ones which do not taper off to fine points, and resemble very much the large one figured by us, as seen in the posterior part of the lateral column in the cervical region.

Explanation of Figures.—Figures 1 to 11. Sections from various regions of both spinal cords, showing the various distribution of the sclerosed patches. Verick No. 2. Eye piece 3.

Fig. 12. A neuroglia cell in the white matter of the spinal cord; increase in the size of the nucleus, with an enormous increase of the protoplasm around it. 1st stage.

Fig. 13. From a section through the corpus striatum; a large number of branching cells are seen distributed in a somewhat coarse mesh of neuroglia fibres; one or two cells proper to the corpus striatum undergoing degeneration. 2d stage.

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* Boll, Die Histiologie und Histiogenese der nervosen Centralorgane, *Archiv für Psych. und Nervenkrankheiten*, Band 4, 1874, p. 1.

† Butzke, studien ueber den feineren Bau der Grosshirnrinde, *Archiv für Psych. und Nervenkrankheiten*, Band 3, 1872, p. 575.

Fig. 14. Section from the brain; axis cylinders without a trace of myeline, lying in a very coarse, loosely arranged neuroglia fibre; a few branching cells showing all stages of degeneration. 3d stage.

Fig. 15. Showing transverse section of artery with hypertrophied muscular coat, and an increase of nuclei in all the coats.

Fig. 16. A very large branching neuroglia cell seen in the white matter of the cord; two nuclei are to be seen, one of which shows a constriction in the middle as if it were about to divide. Verick No. 7. Eye piece No. 8.

A CLINICAL LECTURE ON SYPHILITIC CEREBRAL LESIONS.*

1. Syphilitic Arteritis; 2. Tumor of the Brain.

Gentlemen: The first patient whom I bring before you to-day, I introduce merely for the purpose of exhibiting to you in an exaggerated form a symptom which is also seen in the next patient, who is suffering from some cerebral affection of very obscure origin, and upon whose case I wish to dwell somewhat more at length. This symptom is increased reflex movements; and in the present instance they are certainly phenomenal, being much more marked than in any case which I have as yet had an opportunity of bringing before you. The man had hemiplegia two years ago, and since then reflex muscular action has been most extraordinary. You are probably all familiar with the reflex movement that is caused by striking the leg a sharp blow just below the patella when the limb is flexed and allowed to hang in a relaxed condition; and you perceive how very exaggerated the motion is here when the experiment is tried, the slightest tap, such as would scarcely kill a fly, being all that is necessary to produce it. In the upper extremity the same strongly marked phenomena are exhibited when the tendons of the flexor longis pollicis, biceps, triceps, and other muscles, are thus lightly touched.†

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* Reported by P. Brynberg Porter, M. D. Reprinted from the New York *Medical Journal*, September, 1878.

† This symptom—increased tendon-reflex—has recently been brought into prominence by Profs. Westphal and Erb in Germany, and I have followed their researches with much interest. I had this same patient before you last autumn, and tried the experiment of freezing the skin over the ligamentum patellæ, and then tried the test. The reflex contractions of the quadriceps were then just as well marked as before the freezing, showing that the sensory nerves concerned in the act were not cutaneous but tendinous. It is interesting to note that, since the clinical observation of Westphal, nerves have been discovered in tendons. The meaning of increased tendon-reflex I do not hold to be specially or specifically useful in practice. It simply is one more means of determining increased spinal excitability. Prof. Westphal has claimed that absence of tendon-reflex at the knee

I pass now to the second patient, whose case, I think, we shall find a very interesting and instructive one.

The man's name is Peter R——, an Irishman, thirty-seven years of age. Fifteen months ago he had a chancre, which did not appear until a month after exposure, but which lasted for a considerable time. Six months later, blotches, which did not itch, appeared upon the skin, but no other signs of syphilis became developed. This is not, perhaps, a very satisfactory history of specific infection, but it is, at all events, as clear as we ordinarily obtain from hospital and dispensary patients, and so I think we shall have to accept it as such. With the exception of this eruption, he remained well until September last, when he noticed a weakness of the right leg. There was slowly developing paralysis in this extremity, which continued to increase until about November, since which time there has not been much change in the condition of the limb. The paralysis also affected the upper extremity on the right side, and about a month ago the grasp of the right hand indicated, in three trials, the numbers 35, 35, and 34 respectively upon the scale of the dynamometer, against 40, 40, and 36 respectively marked by that of the left hand. In addition, there has been right facial paralysis and some little impairment of memory. He has never had any injury to the head, nor does he suffer from cephalalgia. There is nothing wrong about the eyes, as far as can be detected by the ophthalmoscope, and the urine has also been examined with a negative result. Finally, speech is entirely normal, there being no evidence of any kind of aphasia. One week ago a new and important symptom showed itself, and that is, weakness of the other lower extremity. There has also now become developed, for the first time, increased reflex. In this instance it affects not only the muscles, but the bladder and other organs in addition ; in other words, it is both tendinous and visceral. Formerly it was erroneously supposed that in such cases there was real paralysis of the bladder, and such is the vague sort of impression still prevalent among many of the profession ; but about twenty years ago, Brown-Séquard demonstrated conclusively that frequent, interrupted, involuntary escape of urine from the bladder was due merely to reflex spasm.

Now let us look for a moment at the objective symptoms. The walk, you perceive, is not that of hemiplegia, lacking entirely the scythe-like leg movement so characteristic of that affection. This man drags his feet after him in a very slow and painful manner ; and while both the limbs are lacking in power, it is evident that the right one is considerably the weaker of the two. In walking he frequently staggers (though there is nothing especially significant about this), and he is unable to stand upon one leg. The naso-labial fold is much more distinct on the left side of the face than on the right, and the left pupil is slightly larger than the right. The tongue is found to deviate somewhat to the left, contrary to the ordinary rule in such cases, according to which it should incline toward the paralyzed side. The relative power

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(knee-phenomenon) is an early symptom of sclerosis of the posterior columns ; and I am pleased to say that observation upon several of my tabetic patients has confirmed this.

in the two hands, as shown by the dynamometer, remains about the same now as it was a month ago. As far as we are able to make out, therefore, the case presents the symptoms of double, incomplete hemiplegia. There is also increased reflex in the muscles, which, though not so extraordinary as in the first case I showed you, is still exceedingly well marked, and, as you perceive, is much stronger upon the right side than on the left, both in the upper and lower extremity.

The next question that arises is, which is the situation of the lesion, or rather lesions, which have produced the phenomena noted? If these had been in the anterior lobes of the brain and near the island of Reil or the third convolution, we should have had some interference with speech; and it is equally certain that they cannot have been in the posterior lobes. The first lesion is, no doubt, to be looked for in the middle portion of the left hemisphere, and the second in the corresponding part of the right hemisphere. I think we can exclude here a lesion of the base near the median line; for, when this occurs, serious trouble ensues much more rapidly than has been the case in the present instance, and the cranial nerves are affected in a much more marked manner.

Now, what is the nature of the lesion? When the nature of the attack that has occurred is taken into consideration, we must undoubtedly exclude both hemorrhage and embolism. As far as relates to the latter, moreover, I may mention that the heart is entirely normal. Again, there does not seem to be any reason to suspect a diffused peri-arteritis, causing aneurisms from which might possibly result the symptoms present in the case. Syphilitic tumors of the brain are quite common; but if there were one at the base here, we should unquestionably have a lesion in the eye, such as choked disk, or neuro-retinitis. Let us, then, inquire whether we may not have here the form of arteritis sometimes met with in syphilis.

Syphilitic arteritis is not degenerative, like atheroma of the vessels. It is true that some authorities are of the opinion that atheroma is proliferative at first. This view lacks proof, however, while there can be no possible doubt that syphilitic arteritis is essentially hyperplastic and proliferative. In the first place, you must remember that the lesion is a diffused one, affecting the arteries in the rest of the body as well as those of the brain. To speak more strictly, it is an endo-arteritis, the hyperplastic formation taking place on the inner surface of the vessel, and usually confined to one side of it. In some cases, however, the whole surface is affected in the same manner, and then the calibre of the artery becomes so diminished by this choking-up process as to finally be almost impervious. The deposit of inflammatory products is not uniform along the whole course of the vessel, but takes place irregularly at various points, so that a number of consecutive little tumors are thus produced. After a time the proliferation cells undergo fatty, but never calcareous, degeneration. By this choking of the arteries the supply of blood to the brain is much diminished, and sometimes we have the same result as occurs in embolism, viz.: parts of the brain become necrosed in consequence. In such cases recovery depends on whether a vital part of the brain has become affected or not. If the third frontal convolution were involved, the patient would never recover his speech, although he might take any amount of iodide of potassium. The prognosis, as you may readily understand, is usually very grave.

In the present case, however, the paralysis is so imperfect that there is room for considerable hope. No essential portion of the brain has probably as yet become affected; but the result, I would impress upon you, is still very uncertain.

This syphilitic arteritis is a very recent discovery in medical science, and it was only in 1873 that Heubner first described it. Since then it has been suggested that we may possibly have a somewhat similar arteritis which is non-syphilitic; but as yet there is not sufficient evidence to prove the point. Heubner says that the specific arteritis is as common as specific tumors of the brain; but whether this is really the fact or not can only be determined by a more extended series of observations than there have as yet been time and opportunity for since the discovery was made.

In the patient now before us we are led to exclude common tumor of the brain on account of the absence of three prominent characteristics of that condition, viz.:

1. Choked disk.
2. Convulsions.
3. Localized pain in the head.

On the other hand, the symptoms correspond perfectly with what we would naturally expect in the syphilitic affection of the cerebral arteries described; and, as there has been a distinct column, the upper part of right column of Burdach, the columns of Goll, posterior part of left anterior column.

Sections just below above in the cervical region (Fig. 9) show the following distribution: the columns of Türck, part of right anterior and lateral column, the entire posterior columns, the periphery of the right anterior horn.

Dorsal region (Fig. 10), columns of Türck, the periphery of both anterior horns; the gray commissure, both lateral columns and both posterior columns.

In the lumbar region (Fig. 11), the columns of Türck, right lateral column, posterior part of left anterior column, entire posterior column, the periphery of both anterior horns. It will be seen from this description that the sclerosed nodules are of various sizes, and occupy the most diverse regions of the cerebro-spinal system, involving the gray as well as the white matter.

Histology.—The histologic study will include the two cases.

For convenience of study and description we will divide the morbid process into three (3) stages. This division, although arbitrary, can nevertheless be observed on a close study of the specimens, and aids very much in a description and the understanding of the various conditions which are observed in the many diseased regions of the cerebro-spinal substance:*

The first stage will comprise the very earliest changes which are to be observed.

The second stage, in which the morbid process has advanced considerably.

The third stage, in which the most extensive changes are to be seen.

The Neuroglia.—First stage. An examination of the neuroglia of the white matter at this stage shows an increase in the size of the nuclei; their number is also slightly increased; there is an increase of the protoplasm around the nuclei, the quantity varying very much in different cells; in most of the cells it is only a little greater than normal, in a few it is enormously increased, as can be seen in Fig. 12, representing two cells seen in the anterior columns of the dorsal region in Case I. The nuclei have not only undergone increase in their size, but they have also assumed the most diverse shapes; in those cells where the protoplasm is very much increased, the nucleus is found at the extreme edge, as if the protoplasm had increased all on one side; we also begin to notice slight processes from the protoplasm; even now there is an appearance of increase in the size of the fasciculi of tissue running between the various areas of nerve tubes. At this time all the surrounding nerve tubes appear to be normal, their axis cylinders and myelinic sheath are perfectly distinct. In the gray matter at this period, the alterations are very similar: a slight increase in the size of the nuclei and the protoplasm around them, and beginning to be visible processes from the protoplasm.

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* The medulla and pons were examined by Dr. Seguin, the two cords and brain by Dr. Shaw.

In the second stage, the nuclei and their surrounding protoplasm have increased in size, their processes are now much larger, and more distinct and apparently more numerous; and there is altogether a much larger number of these branching cells visible. There is now present a diffuse granular appearance; no special fibrillation to be seen; a very close observation shows that the axis cylinders are present, but that their myeline sheaths have gone. (The granular appearance at this period is very probably due to the breaking up of the myelinic sheath into a granular-looking material which becomes diffused among the surrounding tissue, and this perhaps also explains why we do not see the increased fibrillation of the neuroglia.) The process at this period is the same throughout the brain and cord; in the gray as well as in the white matter.

In the third stage these branched cells have grown to an enormous size, their processes are numerous and sometimes of immense thickness and length (Fig. 13); now very little granular material is present, and we see distinctly the enormous increase of neuroglia fibres, and which are of exceeding coarseness; there appears to be no evidence of anastomosis between them. In a stage a little advanced of this (Fig. 14), these branched cells appear much less numerous, they are less distinct, their nuclei are less sharply stained, their outlines are not so perfect, and they sometimes appear as imperfect bodies; they in their turn appear to have lived their day, and are now undergoing decay.

Nerve Fibres.—In the second of the artificial periods which we have created for convenience, we see the first changes in the nerve fibres; their myeline sheath is undergoing disorganization; but their axis cylinders still remain.

In the third period, when the increase of neuroglia fibres is

These two cases form a very interesting study when taken in connection with each other; and in order to bring them more clearly together before you, permit me to once more run briefly over the prominent points in the former one: The patient, a male, and thirty-seven years of age. Fifteen months ago he had constitutional syphilis, and six months ago right hemiplegia, gradually developed, and without aphasia or sensory disturbance. Recently there has been double hemiplegia, the left side being also affected, and with this, increased reflex. Finally, there has been no localized headache, no epileptiform seizures, and no lesion of the optic nerves. The diagnosis is syphilitic arteritis, and consequent localized cerebral softening.

The prognosis in the two cases is very much the same, but probably somewhat better in that of the patient with the tumor than that of the one with arteritis. As regards the case of tumor, however, it is necessary to make the prognosis concerning the affection of sight separate from that in regard to the general condition; for there is great reason to apprehend irreparable atrophy of the optic nerve. It is possible that the tumor may not prove fatal to the patient, though in a considerable number of cases such is the result. This case illustrates very admirably the utility of the ophthalmoscope in the study of nervous diseases, and we are now called upon quite frequently to resort to it in troubles about the head. As instances, I may mention the cases of basilar meningitis occurring in children which I brought before you some little time ago, and in which it would have been quite impossible to make a correct diagnosis without the aid of this instrument. So, too, in Bright's disease and other affections, the instrument is often of the greatest assistance to the general practitioner, as well as to the specialist; and I think that every medical man should be more or less familiar with its use.

LECTURES ON THE LOCALIZATION OF SPINAL AND CEREBRAL DISEASES.*

LECTURE I.

SUMMARY—INTRODUCTORY REMARKS—HISTORICAL CONSIDERATIONS CONCERNING LOCALIZATIONS IN THE SPINAL CORD—PHYSIOLOGICAL ANATOMY, AND PHYSIOLOGY OF THE SPINAL CORD.

GENTLEMEN:—The Faculty having again done me the honor to assign to me some of the extra Thursday Lectures this winter, I have chosen as a subject the very practical question of the Localization of Diseases in the Spinal Cord and Brain.

This topic is now engaging the attention of many of the best minds in the profession, and it is being made the object of careful observation and ardent controversy. Well-reported cases bearing on the question abound in the current medical literature.

The subject has two principal aspects. One of these, that relating to the doctrine of the localization of functions in the brain and spinal cord, is more especially interesting to the physiologist and psychologist. The other aspect, that which concerns us as practical physicians, is with reference to the possibility of making an accurate diagnosis of the seat of the lesion in organic diseases of the nervous centres.

It is this second aspect of the question which I shall discuss with you; and I shall endeavor to do it in as concise and practical a manner as possible—in such a way, in short, as shall enable you to utilize in your future daily practice the various principles and propositions which I shall have the pleasure of presenting to you.

In other words, the following lectures will be upon the rational diagnosis of cerebral and spinal diseases.

In practice, when we have completed the examination of a patient, several questions are put to us by the patient, by his friends, or by ourselves. These are in chronological order: Is there disease? Where is the disease? What is the disease? What are we to do for the cure of the disease or for the relief of the patient? Will the patient die or recover?

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* Delivered at the College of Physicians and Surgeons during the months of December, 1877, and January, 1878. Reprinted from the *Medical Record*, vols. xiii. and xiv., 1878.

Of these questions the one which our client and the world at large consider the most important, is the fourth—that relating to treatment and cure. This preference is natural, but highly unscientific; it is a manifestation of that untrained mental action which demands results and scorns methods, which welcomes empirical achievements (provided they be agreeable), and which conduces to the perpetuation of quackery of all kinds. But to the physician who is not a mere prescription-writer, who aims at infusing as much science into his practice as possible, and who believes that he is not in the world for the purpose of gratifying his patients at so much per visit, but that he owes himself a debt of training and self-culture, and who has a sincere regard for science—to such a physician the first three questions assume a justly great importance. Pray observe that I do not say paramount importance, but great importance. And the superiority of the humanitarian over the scientific duty becomes less glaring if we bear in mind the truth—and I firmly believe it to be such—that success in treatment now depends, and in the future will still more closely depend, upon the scientific study of the human subject in health and disease. In other words, I would impress you with my own conviction that the best trained and most scientific physician, if he be not a closet-student and theorizer, is the best practitioner.

We occasionally hear of an over-fine diagnosis, of extreme caution in the treatment of disease, and of the sweeping application of physiological laws to practice by men who are said to be "too scientific"; but who can number the errors, nay the sacrifices of life, which must be laid at the door of the falsely so-called "practical men," who despise learning and scientific methods? Those of us who see something of the rarer and more formidable kinds of disease fully realize that in medicine, as probably in other applicable sciences, ignorance leads to rashness and crudity in practice, while ripe knowledge conduces to success, or, at any rate, to caution in prognosis and expectancy in treatment.

Of the three diagnostic questions: Is there disease? What is the disease? Where is the disease? the second is the one which forms the key-note of these lectures. Where is the lesion producing the disordered actions or symptoms? The method to be followed in arriving at the solution of this question varies somewhat in different departments of medicine. Some lesions can be seen by the trained unaided eye, or felt by the skilled hand; the seat of others can be determined by auscultation and percussion, by the aid of instruments, such as the ophthalmoscope, laryngoscope, speculum, etc. But in the study of the nervous system greater difficulties are met with; we are, to a great extent, deprived of these physical aids; we cannot appreciate the condition of the brain and spinal cord directly by our special senses, but only by a proper interpretation of the way in which the functions of these parts are performed. In other words, the diagnosis must be made chiefly by reasoning.

What are the conditions or data necessary for correct reasoning in nervous pathology? An enumeration of these will be a brief statement of the way in which I purpose treating the questions before us.

First, you should possess a knowledge of the physiological anatomy of the parts concerned, viz., the brain and spinal cord. You are not obliged, for this purpose, to know much of the histology of the nervous tissue, but you should understand the arrangement of its various parts as recently revealed to us by perfected anatomy and embryology.

Second, you must be well versed in the mode of life and action, or physiology of the cerebro-spinal axis. You must understand, as well as the present state of science allows, what parts are excitable and what inexcitable, which transmit impulses and sensations, which receive impressions, which originate the motor impulse, and which are endowed with special functions.

Third, you need a thorough understanding of the perverted functions of the nervous system, and of other systems connected with it—*i.e.*, of the symptoms of nervous disease; you should cultivate semeiology. And it is here more especially that previous general medical training is of great aid to the student of nervous pathology.

Fourth, you must have a clear conception of the empirical knowledge already gained by numerous post-mortem examinations of persons who have died with disease in the nervous system. You should not accept every proffered autopsy, but critically analyze before making use of it. You may demand that it shall approximate a physiological experiment in exactness and in simplicity.

Fifth, and not least, it is necessary that you have and use a keen critical and logical sense in the appreciation and combination of the above normal and morbid phenomena, in order that you may arrive at sound inductions.

The importance of the combination of the above notions for the study of nervous diseases is immense. Any hypothesis, to be acceptable, must be based upon anatomy, physiology, semeiology, and pathological anatomy. The various crude theories which have reigned awhile in medicine were such as did not fulfill this requirement; some of them were deductions from anatomical data, others applications *a priori* of physiological laws to medicine, others still based solely upon clinical studies, or upon autopsies. Indeed an effort is now being made to break down the growing doctrine of localization of lesions and functions in the brain by just such a one-sided argument. It is claimed by a high authority that facts of the fourth category (post-mortem examinations) contradict, in a perfectly overwhelming manner, the doctrine in question. Now, I trust that, however feebly I may handle the subject, I shall yet be able at the proper time to give you good reasons why we must decline to accept and apply that authority's facts as he does.

Of the few who now deny that we can accurately localize disease in small parts of the brain during the life of the patient, I would, finally, make this critical remark: Their seemingly crushing argument is based upon what seems to me a fundamental error in the appreciation of natural phenomena, and that is, not making allowance for variability and mutability in the highly organized human frame. We now, since the labors of Darwin more particularly, admit that species in the animal and vegetable kingdoms are not fixed forms, but that they may pass into one another by almost infinitely numerous and delicately graded varieties; we know that the ultimate composition of high organic bodies (proximate principles like albumen) varies somewhat; we are prepared to occasionally find (passing to the human organism) the viscera transposed, or to meet with an exanthematous fever without its rash, with a pneumonia unaccompanied by expectorations, or with a painless peritonitis, etc.; in other words, we are, as naturalists and physicians, ready to admit variability and irregularity in the organism. Yet in spite of all this general and special knowledge, the opponents of localization maintain that there can be no irregularity in cerebral action, and they say, by implication at least, that the decussation of the anterior pyramids must always take place, and be total. They demand of those who believe in localization that they should be able to make *every* observed case harmonize with their generalizations. Is this reasoning fit to be applied to natural history? Are we prepared to make use of the mathematical method in pathology?

I shall treat, first, of localizations in the spinal cord and medulla oblongata; and, second, of localizations in the brain proper.

I take up the spinal cord and medulla first because the phenomena are there more simple, and there is less controversy about them than there is with reference to the brain.

HISTORICAL CONSIDERATIONS.

The now voluminous literature of diseases of the spinal cord does not teach us much with respect to the localization of its diseases. The writers of the end of the last century and of the first quarter of this—Frank, Sauvages, Rachetti, Abercrombie, Ollivier—adopted a pathological classification, which has been generally followed in systematic treatises since. Frank recognized, in a pretentious section of his great work on medicine, only spinal neuralgia (rachialgia), myelitis and spinitis (rachialgitis), and hydrorachis. Ollivier, in his last edition, 1837, enlarges greatly upon this primitive list, and describes at least eleven morbid conditions of the spinal cord and its membranes. Brown-Séquard, in 1861, besides demonstrating, as he thought, the existence of reflex paraplegia, treats of all diseases of the spinal cord briefly, and makes an attempt at localizing lesions. He admirably points out how we can diagnosticate a lesion occupying one-half of the spinal cord (hemi-paraplegia and spinal hemiplegia), and also how the height of a lesion in the cord may be estimated. Besides he makes the first attempt at localizing disease in one of the columns of the cord, saying that when the anterior columns are alone inflamed there is paralysis without anæsthesia and little dysæsthesia. He (not knowing of Türck's researches) doubts the pathological independence of locomotor ataxia.

In 1864 appeared Jaccoud's excellent book, which chiefly treats of semeiology and ætiology of spinal diseases. He makes an advance upon previous writers, by considering the question of diagnosis of location of the lesion quite fully, and reaching the following conclusions: 1. Disease in the antero-lateral columns produces palsy without alteration of sensibility; 2. There are no symptoms clearly indicating disease of the anterior gray matter alone; 3. It is easy to recognize if the æsthesodic tract is diseased (sclerosis of the posterior columns) by pain, increased reflex,* and anæsthesia; 4. Lesion in one-half of the cord low down is indicated by hemi-paraplegia, high up by spinal hemiplegia. Leyden, writing in 1874–6, adopts the usual pathological classification, and his work is an admirable treatise. He in numerous places refers to the localization of lesions in the posterior columns, the lateral columns, the anterior cornua, the centre of the cord, and the nuclei of the medulla oblongata, pointing out the diagnosis of each. Hammond, 1876, gives a good *résumé* of the state of knowledge on the subject, basing his classification in part upon notions of localization.

But it is to monographs that we owe most in this branch of pathology.

In 1851 Türck demonstrated the extent and exact distribution of descending degeneration in the spinal cord secondary to cerebral lesions, and in 1857 he found the lesion in locomotor ataxia (then called tabes dorsalis) to be sclerosis of the posterior columns; Dr. J. Lockhart Clarke discovered the lesion of progressive muscular atrophy in 1861-2; Prévost that of infantile spinal paralysis in 1865; Prof. Charcot that of progressive labio-glosso-laryngeal paralysis in 1868; in 1865 the same observer published an autopsy of a case of sclerosis of the lateral columns (three others had been published by Türck in 1856), and in 1875 Prof. Erb, of Heidelberg, and Prof. Charcot delineated the clinical features of this disease—spasmodic tabes, or spastic spinal paralysis. In 1874 Charcot described a mixed type, in which disease of the anterior gray matter is combined with sclerosis of the antero-lateral column—amyotrophic lateral sclerosis. Disease in the central gray matter has been well studied by Hallopeau 1869-70, and Schüppel, and by Leyden, 1876. During the present year Prof. Flechsig has begun the publication of a series of papers upon the systematic diseases of the spinal cord, a work of the greatest merit, based in greater part upon the author's own pathological observations, and upon his yet more important embryological and microscopical studies upon the structure of the spinal cord (1873-6).

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* This word is used in a substantive sense, in imitation of German writers, to designate reflex movements. I think that the word is now much used orally by clinical teachers in this way.

As regards the diagnosis of the location of non-systematic lesions (focal lesions) of the spinal cord we have made no material advance upon the data given us by Brown-Séquard in 1861.

Let us now briefly review so much of the anatomy of the spinal cord as is indispensable to the study of the localization of its functions and lesions. I shall take it for granted that you are acquainted with the usual descriptive anatomy of the nervous centres, and call your attention chiefly to their physiological anatomy.

The spinal cord is a mass of white and gray nervous matter disposed lengthwise in columnar form, and varying in relative proportions at various points. This finer arrangement of the gray and white columns is best studied in transverse sections made at different heights in the spinal cord—for example, through its upper cervical part, through the cervical enlargement, through the lower dorsal region, through the middle of the lumbar enlargement, and near the end of the cord. In every such section we find the same parts, white and gray, but the shape of each is very different on the various surfaces. In general terms the gray substance, or vesicular neurine, is disposed in the centre of the section in the shape of an irregular letter H, with clubbed ends forward. These ends, anterior and posterior, are called horns; they are symmetrical on either side of one section, and the posterior reach out to the very periphery of the cord; dividing the inclosing white substance of each half of the section into two parts, the posterior column and the anterolateral column. By means of pathological study, by histology, and more especially by means of embryology, these white columns have been much more subdivided. Following the latest arrangement by Flechsig, we find in each lateral half of a transverse section of the spinal cord the following parts, proceeding from behind forward:

1. A small triangular column of varying size, lying next to the posterior median septum, in contact with its fellow of the opposite side, the posterior median column, or column of Goll.

2. Externally to this, lying between it and the inner margin of the posterior gray horn, is a broad band containing the sensory fibres of the posterior roots, the posterior root zone, or *zone radiculaire postérieure* (Charcot), or column of Burdach. These two constitute the posterior column of the simpler classification.

3. A small zone lying next to the periphery of the cord, just anterior to the apex of the posterior horn—the direct cerebellar column.

4. Between that and the body of the posterior horn lies an ovoid mass of fibres, the crossed pyramidal column—derived from the opposite cerebral hemisphere by way of the anterior pyramid.

5. Anteriorly to these two, occupying the sides of the section and reaching inward to the gray matter, we find the lateral columns.

6. Lying in front of the anterior gray horns, and extending forward to the periphery, is the *zone radiculaire antérieure* (Charcot), or anterior fundamental column.

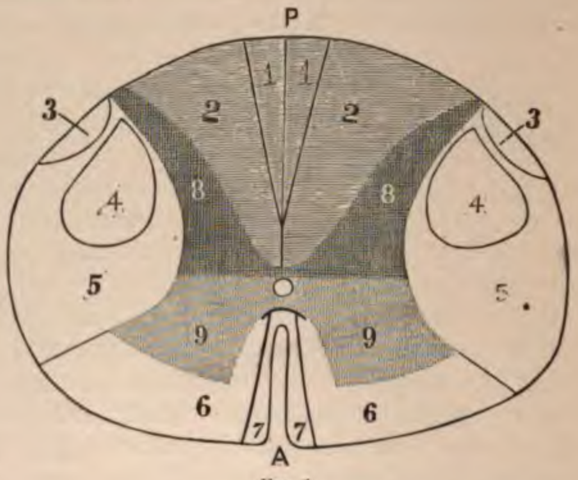

FIG. 1.

Transverse section of the spinal cord.—A. Anterior median fissure; P. Posterior median septum; 1. Columns of Goll; 2. Columns of Burdach; 3. Direct cerebellar column; 4. Crossed pyramidal column; 5. Lateral column; 6. Anterior fundamental column; 7. Direct pyramidal column: 8. Posterior gray horns; 9. Anterior gray horns. Stippled part = gray matter. Shaded part = æsthesodic system. Unshaded part = kinesodic system.

7. A strip of white matter lying on the margin of the anterior median fissure, extending quite to its bottom, is the column of Türck, or better, the direct pyramidal column—derived from the

cerebral hemisphere of the same side, by way of the anterior pyramid.

The simple division of the gray matter into 8, the posterior horn, 9, the anterior horn, will suffice for our purpose. In the accompanying wood-cut the above subdivisions are indicated by numbered spaces.

In an equally aphoristic manner allow me to recall to you the chief physiological attributes of the spinal cord :

1. It is an organ for conduction. Conduction takes place in two directions ; centrifugally for motor impulses, and centripetally for sensory impressions. The paths (I will not say fibres) for sensory impressions ascend only a very small distance in the posterior columns (columns of Burdach) before they enter the gray matter, and there at once pass over to the opposite half of the cord (in man at least). Consequently we say that the conductors of sensation decussate in the spinal cord throughout its whole extent. Motor paths, on the contrary, in the spinal cord proper, remain in one-half of the organ white and gray matters ; they have already decussated (in part) at the crossing of the pyramids. A strange fact to be borne in mind is that very little gray matter may suffice to transmit all sensations. The illustration (Fig. 2) represents, after Brown-Séquard, the course of sensory and motor conductors.

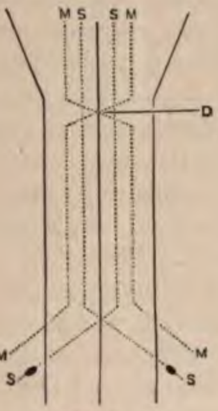

FIG. 2.

Course of motor and sensory paths in the spinal cord, after Brown-Séquard.—D. Decussation of pyramids ; M. Motor paths ; S. Sensory paths.

2. The excitability of various parts of the spinal cord is a point of some interest. It has been quite well settled that no

part of the healthy spinal cord is excitable, except the posterior columns; though very lately Professor Vulpian has discovered traces of excitability in the internal part of the anterior column. However, in morbid states, a great change occurs, and even the unquestionably inexcitable gray substance becomes excitable, giving rise to various morbid sensations and to spasm.

3. The spinal cord has an autonomy of its own, giving rise to reflex motor impulses, and producing others spontaneously. It is also probable that sensory impressions are rendered more perfect in the spinal gray matter, but I am indisposed to attribute consciousness to it. This organ furthermore presides over (executes) many automatic acts, many of them highly complex; such as walking, swimming, standing, to a certain extent eating, dressing, playing upon musical instruments, etc. Part of the spinal gray matter is slowly and painfully educated to perform these actions, and can afterwards do them without any marked cerebral interventions.

4. Besides, the spinal gray matter is supposed to possess a trophic function, to preside over the nutrition of muscles and other tissues, partly through the blood-vessels, and partly directly. This question is under discussion, but the fact remains that disease of the anterior gray matter produces marked atrophy of muscles, and may cause joint-lesions. The spinal cord is also said to embrace several so-called centres; a genital or genito-urinary centre in the lumbo-dorsal region, a cilio-spinal centre in the lower cervical region, and various subordinate vaso-motor regions; of these I recognize only one as useful in such a study as the one we are beginning, viz., the cilio-spinal centre (Budge, Waller, Brown-Séquard). There is a part of the spinal cord (in each of its halves), extending from the fifth cervical vertebra to the second dorsal vertebra, which contains vaso-motor nerve-fibres for the corresponding side of the neck, face, and eye. It also contains fibres whose normal action is to cause a dilatation of the pupil. In estimating the height of a lesion in the spinal cord a knowledge of the location of this cilio-spinal centre is of real utility.

To sum up the physiology and anatomy of the spinal cord, I may divide its section-surface into two great territories, as indicated in Fig. 1. The larger part not shaded, embracing the anterior horns and all the antero-lateral columns, may be designated as the kinesodic system, and the smaller shaded portion, including the posterior horns and the posterior columns, as the æsthesodic system. As the names imply, the latter system conveys and receives sensory impressions, while the former transmits and originates motor impulses, and possibly is trophic in function.

In the next lecture I shall systematically treat of the localization of disease in these physiological and anatomical subdivisions of the spinal cord; first in the two systems, and second, in each column or horn within each system.

LECTURE II.

SYNOPSIS.—SYSTEMATIC LESIONS OF THE SPINAL CORD; LESIONS IN THE ÆSTHESODIC SYSTEM, LESIONS IN THE KINESODIC SYSTEM.

GENTLEMEN:—As a basis for our study of the diagnosis of the location of lesions in the spinal cord, I offer you the following classification, which I think embraces all that sound clinical observation and post-mortem examination will justify us in diagnosticating with certainty.

I. Systematic Lesions of the Spinal Cord.

II. Non-systematic or Focal Lesions of the Spinal Cord.

By the former we are to understand pathological changes which involve one of the gray or white columns of the cord for a part or the whole of its extent up and down, without extension to adjacent columns. Such lesions are almost always symmetrical in the two halves of the organ; and occasionally more than one such lesion may be present.

By the second form of lesion we understand a focus of disorganization or new tissue growth involving the spinal cord in a limited part vertically, and invading diverse columns, or even systems, transversely. These are the *lésions en foyer* of French authors.

The first group may be subdivided as follows:

I. Systematic Lesions of the Spinal Cord.
  a. Lesions in the Æsthesodic System.
    1. Sclerosis of the Columns of Goll.
    2. Sclerosis of the Columns of Burdach.
  b. Lesions in the Kinesodic System.
    1. Sclerosis of the Anterior Columns.
    2. Sclerosis of the Lateral Columns.

a, with changes in Anterior Horns.
3. Degeneration of the Postero-lateral Columns.
4. Myelitis of the Anterior Horns.
5. Degeneration of Ganglion Cells of Anterior Horns.
6. Central Myelitis.

In studying the above forms of disease, I shall apply more or less rigidly the following method:

First; state the general symptoms which indicate disease in the part of the spinal cord (the system) affected. Second; accurately locate the lesion, and state the symptoms produced by it particularly. Third; say a few words concerning the disease in question.

a. Lesions in the æsthesodic system are characterized by the following symptoms: Pain, usually of a peculiar kind, hyperæsthesia, numbness, and anæsthesia; by a peculiar disorder in voluntary movements, viz.: ataxia; and, negatively, by the absence of true paralysis or spasm in the affected limbs.

1. Sclerosis of the columns of Goll, or the posterior median columns. Whether in the ascending secondary degeneration, or idiopathically produced (one case), the lesion occupies the more or less exactly triangular space lying between the columns of Burdach. At the lowest part of the cord the lesion is hardly visible, owing to the smallness of the columns at this point, but in the cervical region it is quite large and distinct. At the calamus scriptorius the lesion disappears, and so far has not been traced higher up. In Pierret's case these columns were sclerosed throughout their whole extent. In cases of ascending secondary degeneration they are affected only above the focus of disease.

The common lesion of the columns of Goll (secondary degeneration) gives rise, so far as we now know, to no symptoms; consequently, we can only infer its presence by determining the existence of a lesion capable of producing ascending and descending degeneration in the spinal cord.

The symptoms in the single case of idiopathic disease of these columns (Pierret's) are too uncertain and too badly reported to be of any use. They consisted in numbness, slight anæsthesia, and a tendency to retropulsion.

It is right to conclude that we cannot to-day directly diagnosticate disease limited to the columns of Goll.

2. Sclerosis of the columns of Burdach, or the posterior root-zones. The lesion begins in the outermost portion of these

columns, near the inner margin of the posterior horns, and, extending forward and inward, ultimately occupies the whole of the columns of Burdach, as shown in the accompanying diagram.

The section showing this lesion, which I now pass around, was taken from the cervical enlargement of the spinal cord of a woman who had typical locomotor ataxia in the lower and upper extremities, gastric crises, diplopia, and amblyopia. In most fully developed cases of locomotor ataxia the sclerosis is found to occupy the posterior median columns as well, *i.e.*, the whole of the posterior columns are degenerated; but, since the researches of Pierret upon the functions and pathological anatomy of the posterior median columns, we must look upon changes in them as secondary to the sclerosis of the columns of Burdach.

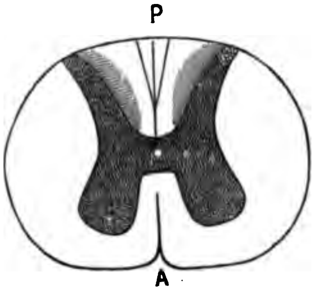

FIG. 3.—LOCATION OF ESSENTIAL SCLEROSIS IN LOCOMOTOR ATAXIA.

In complicated cases of sclerosis of the posterior columns there may also be sclerosis of the lateral columns, or degenerative changes in the central and anterior gray matter.

The symptoms which are characteristic of sclerosis of the columns of Burdach are, in order of importance, pain, hyperæsthesia, anæthesia, ataxic movements. I can only, in these lectures, call your attention to the great diagnostic symptoms, and must not attempt to delineate the semeiology, etc., of any disease; yet the above symptoms deserve more than a mere mention. The pains are peculiarly characteristic—nay, almost pathognomonic. They are of several varieties. First, sharp, lancinating cutaneous pains, appearing in spots, usually circular or oval, in size from one to two inches in diameter, in any part of the limbs which are afterward to become ataxic. A few shootings may take place in one spot, or it may be the seat of pain for twelve or twenty-four hours. A capital point is the capriciousness with which the pains appear in any and all parts of the limbs—toes, thighs, calves, shins, etc. They are vagrant or vagabond pains, in sharp contrast to the fixed *neural* pain of neuralgia. Second, deep, boring pains; and third, tearing or bruising deeper pains, affecting the articulations as well. These pains are also vagabond and capricious in their mode of appearance. They may exist for years without anæsthesia and ataxia showing themselves, and both patient and physician are apt to speak of the pains as "rheumatic," instead of as pathognomonic of an incurable disease. Hyperæsthesia accompanies and succeeds the pain, more especially in the patches of skin which are the seat of stabbing pains. During the paroxysm, and for hours afterward, the patch or patches are exquisitely sensitive, and hyperalgesia exists. Anæsthesia is said to be present very early in the disease, as well as later. In the former case it occupies patches of skin on the legs, arms, and trunk, according to the extension of the disease. In the second case it is found under the feet, later in the legs, and may involve the whole of the lower (and upper) extremities toward the close of the disease.

The ataxic walk consists in a jerky, stamping gait, the abductors and extensors acting over-forcibly; the want of coordination affects large muscular groups, thus differing from chorea, paralysis agitans, etc. But a somewhat similar ataxia may be present in cases of intra-cranial disease, so that I would urge you to fall back upon the sensory symptoms for your diagnosis.

Many practitioners employ what they consider a sure and ready test for locomotor ataxia, viz., they bid the patient try to stand with his eyes closed. If he stagger or fall, he is said to have the disease. Now, I would have you all bear in mind that this test is worthless. Oscillating with closed eyes is a symptom common to many cerebral, spinal, and peripheral morbid states, as hysteria, myelitis, etc. I once artificially froze the soles of a patient's feet, and showed him in this amphitheatre with an excellent stagger when his eyes were closed. He staggered because his feet were anæsthetic, and that is one reason why hysterical women and the bearers of myelitis do the same. We may safely put it in this way: patients with locomotor ataxia, at a certain stage, do stagger when their eyes are closed; but, as the bearers of several other spinal diseases do the same, the sign has no special value.

For many years, and even long after the patient is absolutely unable to stand, there is almost no paresis and no diminution of reflex or of electrical excitability in the affected extremities, unless the sclérosis extend forward into the gray matter.

We ought, I think, to be able to correctly diagnosticate this lesion in its first stage, viz., when only pain and hyperæsthesia are present.

Historical Considerations.—The clinical history of locomotor ataxia has been fairly well known since 1835 or 1840; German physicians describing it in great part under the name of Tabes Dorsalis. Romberg, in 1853 (and earlier), gave almost a perfect picture of the disease; but it was reserved for Duchenne (1859–60) to recognize the value of the pains and of the ataxia, as distinguished from paresis, as cardinal symptoms. Although Duchenne's description of the affection has hardly been improved upon, it must be admitted that he knew nothing of its pathological anatomy, and was quite wrong in his explanation of the symptoms. Yet the lesion of locomotor ataxia, or tabes dorsalis, had already been discovered by Türck in 1857. Unfortunately, he buried his remarkable paper in the Transactions of the Academy of Vienna; and Gull (Sir William) independently made the same discovery in 1859. In France, Bourdon worked out the pathological anatomy in 1861, and since, our knowledge upon the point has grown enormously. The anomalies of locomotor ataxia, both semeiological and pathological, have been best studied by Charcot and his pupils. In 1873, Charcot and Pierret published their cases demonstrating that the primary essential lesion of locomotor ataxia involves the columns of Burdach.

b. Lesions in the kinesodic system are characterized by the following symptoms: Paresis or paralysis, spasm, and muscular atrophy; and, negatively, by the absence of anæsthesia, or of marked and permanent pain or numbness.

1. Sclerosis of the true anterior columns, or columns of Türck, or, best, the direct pyramidal fasciculi, has been known pathologically since the publication of Türck's first papers in 1851. Its semeiology is, however, unknown. We can infer its existence in those cases in which we diagnosticate lesion No. 3 (*infra*), because pathological anatomy shows us that the two lesions are usually simultaneous.

2. Sclerosis of the lateral columns. The lesion consists in an

increase of the neuroglia and atrophy of nerve-fibres in the lateral masses of white substance throughout a greater part or the whole of the spinal cord. It may exist alone as a primary lesion or may be associated with other (secondary) morbid processes, such as No. 4 (*infra*).

The symptoms consist in progressive paresis of the lower limbs, and later of the upper, with increased reflex, and a tetanoid state of the extremities. There is very little sensory disturbance, never anæsthesia; the paralyzed and tetanized muscles do not undergo atrophy; and the bladder is not in the true sense of the word paralyzed.

These symptoms have been designated by various names. Erb calls the symptom-group spastic spinal paralysis; Charcot, spasmodic tabes; and I would suggest the term tetanoid paraplegia or paralysis. Erb in 1875, and Charcot in 1876, Erb again this year, have fully described the clinical aspects of the disease.

In 1873 I described, under the title of tetanoid pseudo-paraplegia, the symptoms of the semi-developed disease, of that transition period when the patient is still able to get about upon his morbidly stiffened limbs. I failed, however, to seize upon the whole clinical picture.

The pathological anatomy of this affection does not as yet rest upon a firm basis. Türck in 1859 published three autopsies in cases of this sort, but their clinical features had not been worked out. In 1865 Charcot reported sclerosis of the lateral columns as the lesion found in a woman who had suffered from aggravated hysteria, with contractures of the extremities, for many years. In the essays of 1876–7 no other autopsies are recorded.

2 *a*. Combination of lesion of the anterior horns with sclerosis of the lateral columns. Charcot, in 1874, first called attention to this complex systematic lesion, and designated the disease as amyotrophic lateral sclerosis.

The cervical enlargement of the spinal cord being nearly always the first part affected, we observe that the symptoms appear first in the hands; a paralytic atrophy setting in with considerable rapidity. Formication and fibrillary movements may be present. The atrophy resembles more that observed in myelitis of the anterior horns (No. 4) than in degeneration of the ganglion cells of the anterior horns (No. 5). In a short time a degree of rigidity appears in the upper extremities, and the legs become first paretic, later, rigid and contractured. This contracture may be greatly relaxed while the patient is in bed, but is exaggerated if he try to stand or walk. In consequence of great atrophy of the interossei, the "claw-hand" deformity may appear. As a rule, the muscles of the lower limbs do not waste. The bladder and rectum are not paralyzed, and no anæsthesia is observed. If the patient's life be prolonged, the disease invades the nuclei of the motor bulbar nerves, and to the above picture we have superadded the symptoms of labio-glosso-laryngeal paralysis.

Cases of this kind had been observed prior to 1874, but it is Charcot who in that year first gave us a clear statement of the pathological anatomy and semeiology of the disease. The connection between the two lesions, disease in the anterior horns, and sclerosis of the lateral columns, is by no means understood, and it may be questioned whether they are pathologically related.*

3. Degeneration of the posterior part of the lateral column, or (better) of the crossed pyramidal fasciculus. The lesion occupies, in a transverse section, a part of the white substance which lies between the lateral column and the posterior gray horn. It is separated from the periphery of the cord by healthy tissue, the direct cerebellar fasciculus. This alteration of tissue is secondary to a lesion in parts of the nervous centres above the decussation of the pyramids; in the anterior pyramids, the great motor tract in the basis cruris cerebri, the anterior part of the internal capsule, the nucleus caudatus, the convolutions comprising the excitable districts of the cerebrum. Lesions of any of these parts cause what is known as secondary degeneration throughout the motor tract, to the lowest part of the spinal cord. Consequently, the lesion in the spinal cord is always (?) on the side opposite to that on which the primary, supra-spinal disease exists. Hence, also, we usually find descending degen-

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* Since this lecture was delivered, Prof. Flechsig, of Leipzig (in *Archiv der Heilkunde*, 1878, Heft 1), has made an elaborate critique of Charcot's cases and others, and claims that the lesion in the lateral columns is chiefly in the postero-lateral columns, as in descending degeneration from cerebral lesion, and that the nature of the lesion seems more like a degenerative than a sclerotic one. Flechsig suggests that future research may reveal a double lesion in the cerebrum, or, at any rate, in the upper part of the intra-cranial motor tract. This view seems to me well worthy of consideration, and a careful attempt should be made to verify it.

eration only in one-half of the cord. It should be remembered, however, that, as a portion of the anterior pyramid of the medulla does not decussate, but descends as the direct pyramidal fasciculus, or column of Türck, we must expect to find in many cases a similar lesion of this fasciculus on the same side as the supra-spinal lesion (No. 1). For example, after a lesion involving the great motor tract in the left hemisphere, we shall find descending degeneration of the crossed pyramidal fasciculus in the right half of the cord, and of the anterior column, or direct pyramidal fasciculus in its left half.

The symptom characteristic of this morbid condition is the secondary contracture, or late contracture, which so often succeeds attacks of hemiplegia, being superadded to paralysis or anæsthesia. This lesion may be complicated with No. 4, when atrophy of some of the paralyzed and rigid muscles supervenes.

In briefly mentioning the historical data connected with this lesion, I must again mention Türck, the great pioneer in the pathology of systematic lesions of the spinal cord. In 1851 and 1853 he exactly described the crossed part of the lesion. An excellent study of this and other forms of secondary degeneration was made by Bouchard in 1866, under the direction of Profs. Vulpian and Charcot. Last year Flechsig published his remarkable researches upon the nervous centres, and more exactly defined the seat of both the crossed and the direct degenerations. To Prof. Bouchard is due the credit of completing the clinical picture, by pointing out the value of the symptom contracture.

LECTURE III.

1. SYSTEMATIC DISEASES OF THE SPINAL CORD, CONTINUED; LESIONS IN THE KINESODIC SYSTEM. 2. NON-SYSTEMATIC OR FOCAL LESIONS OF THE SPINAL CORD; LESIONS AT DIFFERENT HEIGHTS IN THE ORGAN.

GENTLEMEN:—There remain for consideration a few of the systematic lesions of the spinal cord.

4. Myelitis of the anterior horns, or poliomyelitis anterior. Like all affections of the kinesodic tract, this affection is characterized by the predominance of motor symptoms and the absence of sensory ones. But we also meet with great trophic changes—muscular atrophy—in this disease.

The lesion consists, as we know from the autopsies by Gombault, by Cornil and Lépine, and by Déjérine, in an inflammation of the anterior gray horns of the spinal cord, leading to atrophy, and even destruction of the motor (and trophic?) ganglion-cells. The change in the cells is acute pigmentary degeneration.

In those regions of anterior gray matter corresponding to the paralyzed parts, hardly any motor ganglion-cells remain. Other lesions are degenerative changes in the motor nerves, as far as their termination, and muscular atrophy, usually without fatty metamorphosis.

The symptoms of the affection vary in the three varieties: acute, sub-acute, and chronic.

a. In acute febrile poliomyelitis* anterior we observe a sharp, remittent or continued fever, lasting one or several days, accompanied in many cases by pains in the limbs, and sometimes by slight numbness. This is followed, suddenly, as a rule, by extensive paralysis, the fever ceasing. The paralysis may affect all the limbs, or two of them, or one only; it tends to diminish spontaneously to a marked extent. No anæsthesia is present, and but little numbness. Reflex movements are reduced or abolished, the bladder and rectum act normally, and there is no tendency to bed-sore. But other even more characteristic symptoms soon follow. In a few days the nerve-trunks in the severely palsied limbs lose their galvanic and faradic excitability, and the muscles, while ceasing to respond to the faradic current, contract slowly, and with abnormal formula, to the galvanic current—we have the degeneration-reaction. A little later, after two or three weeks, the palsied muscles undergo rapid atrophy, an atrophy which is progressive if no recovery is to take place. Ultimately only one muscular group may remain paralyzed and atrophied.

b. There is a non-febrile acute myelitis anterior. The patient, usually a child, is put to bed well, and awakes in the morning with one or more paralyzed limbs, with the subsequent symptoms as above.

In these two forms are to be ranged nearly all cases of infantile spinal paralysis so-called, and many cases of the same disease occurring in the adult.

c, d. Febrile and non-febrile sub-acute myelitis of the anterior horns differ from the above only in degree of acuteness and in rapidity of development, and need no detailed description.

e. Chronic myelitis of the anterior horns is often mistaken for progressive muscular atrophy; yet a diagnosis, is, I think, frequently possible. Often in this variety of poliomyelitis there occur severe neuralgic pains in the limbs which are to undergo palsy and wasting. These phenomena, when they appear, weeks or months after the first symptoms, attack whole muscular groups at once, and we do not observe the fibrillary or fascicular wasting of progressive muscular atrophy. The reactions are like those found in the acute and sub-acute form. No anæsthesia appears. This rare form has been observed in children and in adults.

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* See p. 84.

It is only very recently that we have had a correct knowledge, clinical and pathological, of myelitis anterior. Prior to 1865, infantile spinal paralysis, though well known clinically (Heine, 1840), was thought to be due to congestion of the spinal cord, etc.; but in that year Prévost, working with Charcot, discovered the lesions in the anterior horns, and since numerous autopsies have yielded the same results. As regards the disease in the adult, it was correctly observed and classified as far back as 1847 (Duchenne), and in subsequent years by Charcot and others. Its pathological anatomy was not discovered until 1873 (Gombault) and 1875 (Cornil and Lépine). Although we need more light upon the intimate nature of the pathological changes occurring in this disease, I believe it to be now quite firmly established in nosology. Its diagnosis should be readily made by all practitioners.

5. Degeneration of the ganglion-cells of the anterior horns. As indicated by the name, the lesion in this affection is degenerative rather than inflammatory. This is true in the sense that changes in the neuroglia are wanting as a rule, and that the molecular death of the ganglion-cells takes place very slowly. This is in marked contrast to the suddenness and extent of the lesion in No. 4. Ganglion-cells are found in every stage of transition from simple increase of normal granular contents to mere roundish masses of granules—granular bodies; in some parts of the anterior horns not even vestiges of cells remain.

The symptoms of this lesion are fibrillary contractions followed by atrophy. Sensory symptoms are wholly wanting as a rule, and in a few cases the wasting limbs are the seat of some neuralgic pains. There is at no time a true paralytic condition, since the loss of power is precisely in proportion to the destruction of muscular tissue.

The mode of occurrence of the atrophy deserves a remark. Whole muscles or muscular groups do not waste away rapidly as in No. 4, but the muscular tissue undergoes change, bundle by bundle, very slowly. It thus happens that we see one or two large fasciculi in a muscle quite atrophied, while the adjacent fasciculi of the same muscle are normal, or only show fibrillary contraction. Several months may elapse before a muscle is wholly atrophied. Another feature of the atrophy is that it attacks by preference certain muscular groups, as those of the hand, chest, thighs, etc. It also affects simultaneously, or nearly so, parts which are symmetrical and homologous. If we examine the wasting muscles with the faradic current, we obtain yet another diagnostic sign: in this affection reaction to the faradic current is lost only in the absolutely atrophied muscles or parts of muscles. This loss of reaction is in direct proportion to the atrophy, whereas in myelitis of the anterior horns whole muscles and muscular groups lose their faradic reaction *en masse*, and this, too, often before any marked degree of wasting has appeared. By the latter test, by the distribution of paralysis and atrophy, and by the predominance of neuralgic pains in chronic myelitis of the anterior horns, we may nearly always distinguish it from progressive muscular atrophy. Of course, the two diseases are congeners, and their pathological relationship may even be closer than we now suspect.

Cruveilhier, Aran, Duchenne, and Roberts admirably described the clinical aspects of this disease, and its naked-eye pathological anatomy. But it is to the very recent labors of Lockhart Clarke (1861–2), and of Charcot and his pupils, that we owe the exact determination of the lesion in the anterior horns of the spinal cord. It is also only within the last ten years that we have clearly distinguished pure muscular atrophy from the various forms of symptomatic atrophy.

6. Central myelitis. · An inflammation of the central parts of the spinal gray matter, involving the æsthesodic and kinesodic tracts, extending in some cases throughout the whole length of the organ. The proliferative changes and exudations result either in the formation of a central plug, or the development of a cavity, which is filled with clear fluid. The anterior horns and the various columns of the spinal cord are more or less involved through extension of inflammatory action, or by compression. As might be inferred from the above, the symptoma-

tology of the affection is obscure and complex. Early in the disease, disorders of sensibility—as numbness, formication and pain, followed by anæsthesia—are prominent. Irregularly distributed paralysis, with or without atrophy, is also present. The diagnosis in this early stage, which may last years, is next to impossible. When the disease is fully formed, we have quite a distinct symptom-group. The arms alone are sometimes paralyzed, atrophied, and anæsthetic, while the legs are the seat of abnormal reflex, even to the degree of tetanoid walk. In other cases we see a general paralysis and universal anæsthesia, with contracture of some muscular groups, paralysis of the bladder, and the appearance of bed-sores. If the cervical enlargement be the seat of diffused central myelitis, pupillary symptoms are seldom wanting, and the pulse is accelerated. The disease is eminently a chronic one, years being required for its full development.

Historical considerations.—Ollivier (1836) observed and described central myelitis with formation of cavities, but not much was learned of the disease and its semiology until Schüppel published his paper (Ueber Hydromyelus) in 1865. Hallopeau, in 1871-2, contributed a series of elaborate articles upon the subject to the *Archives Générales de Médecine*, and in the last few years Westphal and Leyden have paid considerable attention to this rather rare form of disease.

2.—NON-SYSTEMATIC OR FOCAL LESIONS OF THE SPINAL CORD; LESIONS AT DIFFERENT HEIGHTS IN THE ORGAN.

A variety of lesions may involve the whole or a large part of the spinal cord at a given level, extending transversely through its various columns. These are focal lesions, and chief among them we find, injuries of all kinds; compression by bone or by a tumor; transverse sclerosis; transverse softening; hemorrhage in cord; tumor in the cord, etc.

The nature of the lesion is sometimes such (fracture of vertebræ) as to indicate at once the seat of injury to the spinal cord; but in many cases the peculiarity in the symptoms is not due to the nature of the lesion, but to its location high up or low down. The diagnosis of this location is possible only by the aid of anatomical and physiological knowledge.

The following diagram, made from data furnished by Malgaigne, may assist in estimating the height of a lesion. It indicates the point of origin of the important nerves and plexuses, and the seat of so-called centers relatively to the spinous processes of the vertebræ.

In general terms we may say that these focal lesions give rise to paralysis, numbness and anæsthesia, to modifications of the reflex function, usually an increase; that they cause paresis or paralysis of the bladder and the sphincter ani, and that they set up a great liability to bed-sores. This general sketch varies greatly from that of the symptoms of any of the systematic spinal lesions, except No. 6, central myelitis. This great dissemblance is a necessary result of the difference in the location of lesions; focal lesions involving kinesodic, æsthesodic, and trophic parts of the cord, and cutting off the inhibitory action of the encephalon (Setschenow), upon the spinal cord.

I shall attempt to make clear to you the diagnosis of lesions placed (1) in the lower lumbar enlargement; (2) just above the lumbar enlargement; (3) in the mid-dorsal region; (4) in the cervical enlargement, and (5) in the upper cervical region.

1. Focal, transverse lesions in the lower part of the lumbar enlargement. The motor symptoms produced by such a lesion are paralysis of the muscles innervated by the sciatic nerve—those of the feet, legs, posterior aspect of the thigh and the nates. The sphincter ani will be weak or paralyzed; the bladder unaffected. The reflex movements of the paralyzed muscles are reduced in force, or absolutely wanting; wanting if the whole of the gray matter in the lower end of the cord is diseased in such a way as to destroy its functions. Dependent also upon this extent of lesion downward is the state of muscular irritability. If the condition be as just described, the paralyzed muscles undergo atrophy, and lose their faradic contractility. The sensory symptoms are various; at the beginning of the disease (often before paresis) there is numbness in the soles of the feet, without anæsthesia. Later the numbness may appear in the whole foot, the calf, the posterior aspect of the thigh, and actual anæsthesia may supervene. If the soles of the feet lose their sensibility, the patient is no longer able to maintain his equilibrium when his eyes are closed. Another sensory symptom of importance is the cincture or band feeling, which, whether like a cord or like a belt round about the palsied parts, indicates (in accordance with the distribution of sensory nerves) the upper limit of the spinal lesion. Thus, in the condition we now study, the cincture feeling will not be about the waist or groin, but

around the ankle, leg, or thigh. The above symptoms make an incomplete paraplegia.

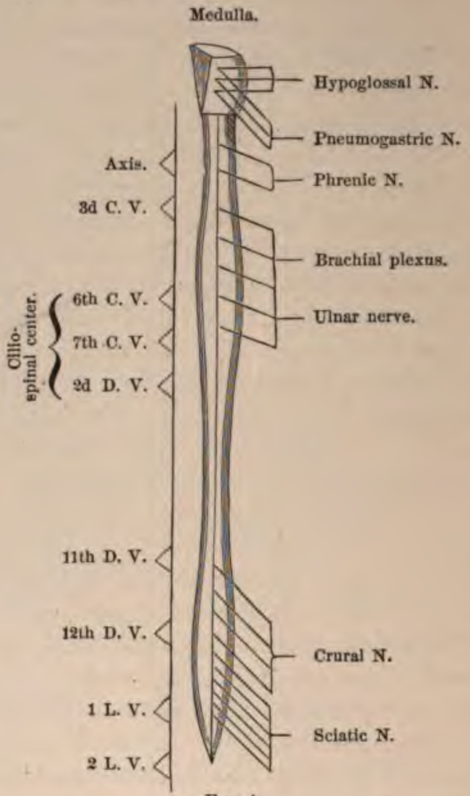

FIG. 4.

Relation of spinous processes of vertebræ to spinal nerves. After Malgaigne *Traité d'Anatomie Chirugicale*, vol. ii., pp. 32-3.

2. Focal, transverse lesions, situated just above the lumbar enlargement. Conduction to and from the brain is interfered with, but the gray matter of the lumber enlargement retains its functional activity in great measure; hence we have a very different semeiology from that described above. In addition to weakness in the feet, there is developed a more or less complete paralysis of the whole of the lower extremities, from the pubis down. The rectum and bladder are paralyzed, the latter showing defective action by slow, interrupted micturition, or by retention, while constipation expresses the rectal paresis. The reflex movements of the paralyzed limbs are usually exagger-

ated, sometimes enormously so. This increase in reflex movements gives rise to the combined tonic and clonic movements of the paralyzed limbs, which Brown-Séquard years ago designated spinal epilepsy. This often seems spontaneous, when the patient is in bed for example, but it is certain that peripheral irritations, whether from the contact of the bed-clothing or from fæces in the rectum, urine in the bladder, etc., always precede and cause it. The preservation and increase of reflex is owing to the continued (increased) activity of the lumbar gray matter below the lesion, and the cessation of cerebral inhibitory action. Dependent upon the former fact also, we observe that the paralyzed muscles do not undergo positive atrophy, and that their electrical reactions are normal or exaggerated. Erections occur, and coition may be accomplished in these cases and in those to be described in the next paragraph.

As regards sensory symptoms we have, as in No. 1, numbness and anæsthesia in the paralyzed parts, extending as high upward as the groins or waist. The cincture feeling is nearly always present, and is placed by the patients round about the body at the level of the hips, or waist just below the umbilicus. In some cases the cincture is incomplete, and the feeling is likened to a firm grip in the patient's side or hip. The above description is a picture of common, complete paraplegia.

3. Focal, transverse lesions in the middle or upper dorsal region. The motor and sensory symptoms produced by a lesion so placed are very similar to those just described, with the following additions. The abnormal reflex movements are often more marked than in No. 2, and the cincture feeling, index of the upper limit of the lesion, is placed at or above the umbilicus, around the lower ribs, or even just under the arms. In this condition the rectum and bladder may after a while partly regain their functions; *i. e.*, their contents are involuntarily expelled from time to time by reflex action.

If the urine dribbles away it is not because the "sphincter" (?) of the bladder is paralyzed, but because there is retention and overflow—a state demanding the daily use of absolutely perfect and carbolized soft catheters. In the earlier stages of the affection, the occurrence of increased reflex action in the paretic limbs gives rise to a tetanoid state during attempts at standing or walking, and too hasty, quasi-involuntary micturition and defecation. Later in the disease the paralyzed limbs may become contractured, by reason of secondary changes in the lateral columns.

4. Focal, transverse lesions in the cervical enlargement. According as the lesion suddenly or gradually affects the whole of the cord transversely, or according as it is placed on the lower or upper portions of the enlargement, somewhat different symptoms are obtained. They always, however, bear a general resemblance to those in Nos. 2 and 3.

a. A partial lesion may for a time produce symptoms, numbness and paresis, in the arms and hands alone, the lower limbs being only weak and showing increased reflex. Later, as the lesion extends, the legs as well as the arms are paralyzed, and the cincture feeling exists high up.

b. A lesion involving the cord at the level of the eighth cervical and first dorsal nerves (see sketch) will give rise to paralysis, often with atrophy and loss of faradic reaction in those muscles of the upper extremities which are animated by the ulnar nerves, *i. e.*, nearly all the small muscles of the hands, and some of the flexors of the wrist and fingers. There will be sensory symptoms in the same district; and the cincture feeling, if present, will be across the upper part of the chest. The lower extremities are paretic or wholly paralyzed, numb, or anæsthetic, according to the completeness of the destruction of the spinal tissue. In severe cases nearly all the intercostal muscles will also be paralyzed, and thus life will be much more jeopardized than by lesions placed lower down. The danger is all the greater because the expiratory muscles (intercostal, triangularis sterni, abdominal muscles) are paralyzed also.

c. If the lesion be situated in the upper part of the enlargement, the motor and sensory symptoms will be apparent in nearly the whole of the upper extremities, as well as below them. The reflex capacity, the state of bladder and rectum, the faradic reactions of muscles remain substantially as in Nos. 2 and 3. The cincture feeling is referred to the level of the clavicles, or a little lower, across the chest and the arms below the deltoid. The difficulty of breathing is even greater than in *b.* The symptom groups produced by lesion No. 4 are often designated by the names of cervical paraplegia, or general paralysis.

In case of lesion in any part of this region (from the level of the fourth dorsal to that of the fourth cervical nerves) there may be pupillary and facial vaso-motor symptoms. If the lesion be of such a nature as to cause irritation of the cilio-spinal centre, the symptoms are dilation of the pupils and pallor of the face, while if there be a loss of the activity of the cilio-spinal centre, the pupils are small and the face and ears flushed and hot. It must be added that these vaso-motor and ciliary symptoms are not by any means as frequently observed as theory and experimentation would lead us to expect.

The same may be said with reference to some peculiarities in the action of the heart and variations in the bodily temperature which have been observed. In severe traumatic lesions in this region, we often find retardation of the pulse, and great elevation of the temperature of paralyzed parts.

Focal, transverse lesions in the upper cervical region. These, like No. 4, produce cervical paraplegia, but a much more complete one. The patient is wholly paralyzed below the head, and the entire body may be anæsthetic. Of necessity the phrenic nerves are paretic or paralyzed, according to the completeness of the injury to the cord, and life is almost immediately terminated by asphyxia. Cases of this category are nearly always of a surgical character; non-traumatic lesions of this region being exceedingly rare. There may be ciliary and facial vaso-motor symptoms here as in No. 4, and the bodily temperature and pulse-rate are variable. Life is preserved too short a time to allow of much study of these symptoms. In slowly developed lesions we may have phenomena of irritation, as hiccough, dyspnœa, acceleration of the pulse, together with paretic symptoms in the arms and chest, later in the legs.

LECTURE IV.

2. NON-SYSTEMATIC OR FOCAL LESIONS OF THE SPINAL CORD CONTINUED; LESIONS INVOLVING ONE LATERAL HALF OF THE SPINAL CORD, IN ITS LOWER AND UPPER REGIONS; DIAGNOSIS OF SPINAL HEMIPLEGIA.—ANATOMY AND DISEASES OF THE MEDULLA OBLONGATA.

GENTLEMEN:—When a focal lesion, caused by spontaneous disease, or by traumatism, involves one lateral half of the spinal cord more or less exactly, we observe striking and characteristic symptoms in the patient. Some of these are in relation to the height of the lesion in the organ as pointed out in the preceding lecture, but the most important ones are dependent upon the fact that one lateral half of the cord is injured in its kinesodic and æsthesodic tracts. A reference to Figures 2 and 4 in preceding lectures will facilitate the comprehension of what I shall have to say upon these lesions.

According as the lateral focal lesion is placed low down or high up in the spinal cord, we denominate the symptom-groups as hemi-paraplegia in the first case, spinal hemiplegia in the second.

a. Hemi-paraplegia. A tumor compressing one lateral half of the spinal cord in its dorsal (case by Charcot) or lumbar regions, a knife-cut, a contusion by a piece of broken vertebra, or a patch of hemorrhage or softening, will give rise to this symptom-group. Let us suppose the lesion to be situated in the right half of the spinal cord. As a result (see Fig. 2) the motor paths from the brain and upper spinal cord to the right lower extremity are cut off, together with the sensory paths which, crossing the median line below the lesion, supply the left lower extremity with sensibility.

In the living human subject we observe motor paralysis, more or less complete in the right lower extremity, and the sensibility of this member is preserved or increased. If the lesion be traumatic and quite complete, hyperæsthesia and increased temperature are present. In the left lower extremity, on the contrary, we find no paralysis, but more or less complete anæsthesia. It is noteworthy that the so-called muscular sense is not abolished in that anæsthetic limb. The bladder and rectum may be paralyzed. In some cases the distribution of symptoms in the two extremities is not as typical and clear as above stated, some paresis appearing on the side opposite the lesion, and slight loss of sensibility existing on the paralyzed side. This, I need hardly say, is owing to the fact that the lesion crosses the median line.

As regards constriction bands, increased reflex, nutrition of muscles, and visceral paralysis, regard must be had to the exact height of the lesion in the cord, as determined in sections 1, 2, 3 of the preceding lecture.

This form of paraplegia can be exactly reproduced in animals. It is now nearly thirty years since Brown-Séquard showed to the Biological Society of Paris, animals (Guinea-pigs, etc.), in which hemi-paraplegia had been produced by cutting one lateral half of the cord in the dorsal region. The operation is not very difficult, and the results are always striking.

b. Spinal hemiplegia. A lateral focal lesion in the cervical enlargement, or above it, will give rise to the following symptoms, more or less exactly distributed. If the right half of the organ is the seat of the lesion, we observe that the arm and leg on the same side are paralyzed, the intercostals usually escaping, while on the opposite side there is no paralysis, but more or less perfect anæsthesia exists to the median line of the body, and as high as the limit of distribution of sensory nerves coming from the spinal cord just below the lesion. The absolute height of the lesion is to be determined as in sections 4 and 5 of Lecture III. In these cases we nearly always find, on the same side as the injury, contraction of the pupil, redness and increased temperature of the face and ear. These symptoms are due to paralysis of the vaso-motor and ciliary centers in the spinal cord. Often the paralyzed limbs are hotter than the anæsthetic.

In some cases, owing to incomplete destruction of one lateral half of the cord, the arms, thorax, and face alone exhibit symptoms, the legs remaining normal. In other cases various degrees of abnormal reflex action are present in the affected lower limbs ; sometimes more on the anæsthetic side.

We owe a clear conception of these interesting forms of spinal paralysis to Brown-Séquard, although some cases of spinal hemiplegia had been placed on record before him (Sir Chas. Bell, Ollivier, Oré, and others). This distinguished physiologist and physician produced spinal hemiplegia and hemi-paraplegia in animals from 1849 on, and in 1863–5 published elaborate memoirs, in which he collected all the cases then known, and critically studied their semeiology. In 1868 and 1869 the same author published other cases and remarks which have placed our knowledge of this subject upon a firm foundation. Some doubt exists as to the absolute correctness of Brown-Séquard's law of the course of motor and sensory paths in the spinal cord in animals, but in man it would seem, from the study of many cases, that the law holds good.

ANATOMY AND DISEASES OF THE MEDULLA OBLONGATA.

Leaving the diseases of the spinal cord to proceed to the consideration of those of the medulla oblongata, I must again refresh your memories upon the physiological anatomy of the parts concerned; mentioning only such data as will be of use to us in our diagnosis of location of disease. The first, and perhaps the most important, point I wish to call your attention to is the decussation of the anterior pyramids at the junction of the spinal cord and medulla. This decussation is made up of bundles of nerve fibers coming from the anterior pyramids of the medulla, crossing the median line, and continuing their way down the cord in each of its halves, constituting that part of the lateral column known as the crossed pyramidal column. Another bundle extends downward, without crossing the median line, into the inner part of the anterior column, the so-called direct pyramidal column (compare Fig. 1, Lecture I.). According to the books, the uncrossed portion of the pyramidal fasciculus, the direct pyramidal column, is much smaller and less important than the other bundle of fibers which cross the median line.

Of immense importance for the study of localizations in the brain, and particularly for the estimation of the symptom hemiplegia, is a knowledge of the exact extent and regularity of this decussation, or more strictly speaking, semi-decussation. If we are to follow text-books we shall be led to believe that the semi-decussation always occurs in about the same proportion on the two sides: a view which Brown-Séquard and others admit while trying to demonstrate that a lesion of the brain may produce paralysis on the same side of the body. Still, in past times a few facts had been recorded against the constancy of the pyramidal decussation, but it was not until Flechsig published his embryological researches in 1876 that it was known how *very variable* is this decussation. Examining the spinal cord of sixty fœtuses, this investigator found that the proportion in the size of the crossed and direct pyramidal columns varied almost infinitely between the following figures: in one case the direct pyramidal column was equal to 90, the crossed column to 10. In another case at the other extreme the proportion was 0 to 100; in other words all the pyramidal fibers had crossed the median line. There was no case in which there was absolutely no decussation, but Flechsig correctly remarks that there is no reason why such a specimen should not be met with. The majority of the fœtuses had an ordinary semi-decussation, the crossed bundle predominating over the direct. The above variations in the pyramidal decussation are diagrammatically represented in Fig. 5. Besides, Flechsig determined that there were other irregularities in the proportionate size of the four columns (two crossed pyramidal columns, two direct) which result from the breaking up of the pyramids, but this is not essential to our present study.

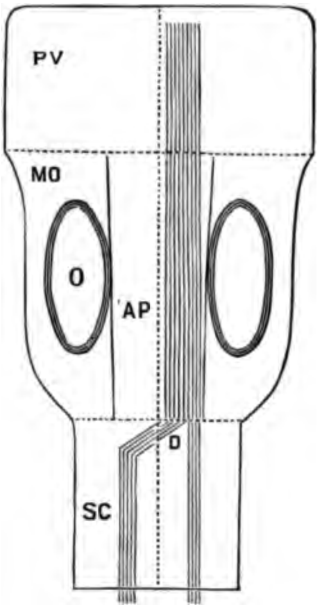

FIG. 5.

Diagram explicative of pyramidal decussation: P V, pons Varolii; M O, medulla oblongata; O, olivary body; A P, anterior pyramid; D, decussation; S C, spinal cord. The direct and crossed bundles vary very much in size, as shown in the following ratios of crossed and direct: 100 : 0, 92 : 8, 84 : 16, 70 : 30, 52 : 48, 35 : 65, 10 : 90.—(*Flechsig.*)

The practical bearing of this discovery is very great, for it will be readily understood that in the exceptional cases in which the pyramidal decussation is nearly wanting, an unilateral lesion of the medulla, pons, or brain must give rise to symptoms of paralysis on the same side as the lesion.

Again, in those few cases in which the semi-decussation is nearly equal, an unilateral lesion above the spinal cord will produce weakness on both sides of the body. Lastly, in the immense majority of cases, those in which most of the pyramidal fibers cross the median line, we obtain the classical crossed hemiplegia. It is ignorance of these researches of Flechsig which makes that distinguished physiologist, Dr. Brown-

Séquard, invert the reasoning and draw chaotic and iconoclastic conclusions respecting the mode of production of hemiplegia (and other symptoms of brain disease). Starting with the generally received doctrine (never proven in a scientific way) that the pyramidal decussation is invariable and nearly total, and finding scattered in medical literature more than three hundred cases in which paralytic symptoms appeared on the same site as the lesion, he concludes that we are all wrong in considering hemiplegia to be due to destruction of or pressure upon motor tracts in the brain and basis cerebri.

Bearing in mind Flechsig's demonstration, the erroneous logic of the above is evident. Besides, these exceptional cases, so laboriously collated, should be looked at in another way. Most of them are old cases, *i.e.*, cases reported before modern cerebral anatomy was understood; many of them are related by men utterly or relatively unknown. Furthermore, any one who has worked much in the post-mortem room will appreciate how easily right is wrongly written, when left is meant, as a brain is turned over and around for examination. Consequently, we might claim the right to reject as worthless many of these three hundred or more cases. Suppose, however, that they are accepted as *bona fide* examples of palsy on the same side as an encephalic lesion, and that we compare them with the thousands of cases of classical hemiplegia on the side opposite the cerebral lesion, do we obtain a proportion greater than that observed in Flechsig's series of fœtuses, one to sixty? I think not.

I have thus digressed from proper medulla diseases in order to treat the subject of pyramidal decussation fully, *i. e.*, anatomically and clinically, in one lecture. Having done this will save much repetition in treating of lesions above this point.

The pyramids whose decussation we have studied are the direct motor tracts which connect the cerebral cortex with the spinal cord. They are externally visible on the anterior (inferior) surface of the medulla, can be traced in the pons and crura, and thence into the internal capsule in the white centre of the hemispheres, to those parts of the cerebral cortex which are now considered as in some way motor. In subsequent lectures I shall describe this great motor tract more fully, and give you the reasons for believing it to be continuous from the cortex to the end of the spinal cord. The remainder of the kinesodic system of the medulla embraces longitudinal bundles of fibres which lie between the two olivary bodies (supposed to be in connection with the ganglia at the base of the cerebrum and the various nuclei of motor nerves in the floor of the fourth ventricle. These nuclei, or groups of motor (and trophic?) cells, represent the anterior horn cells of the spinal cord, strangely thrown backward and toward the median line. They differ from the cell-groups of the anterior horns also in being more differentiated in relation to the nerves which arise from them. Reckoning from below upward, we find the nuclei of origin of the spinal accessory (11th), hypoglossal (12th), facial (7th), and abducens (6th) nerves. The last two lie on the confines of the medulla and pons. These nerves and their nuclei must, I think, be looked upon as active in three ways: 1, by their own (trophic?) neurility; 2, by reflex action set up through the adjacent and correlated sensory nerves; 3, by impulses coming from supra-bulbar parts, great basal ganglia, cortex of the brain. This last connection is undoubtedly a crossed one, *i. e.*, the fibres or paths which transmit the motor impulse down to these nuclei cross the median line at some unknown distance above them. There is therefore another motor decussation besides the great one described above.

The æsthesodic system of the medulla oblongata occupies its lateral portions chiefly, and a part of the gray matter under the floor of the fourth ventricle, outside of the series of motor nuclei. In this gray matter we find, from below upward, the nuclei of origin of pneumogastric (10th), glosso-pharyngeal (9th), and acoustic (8th) nerves. All through the lateral region of the medulla posterior to the olivary bodies lies the great ascending root of the trigeminus (5th) nerve, which meets with groups of cells throughout this region down to the uppermost parts of the spinal cord.

These various nuclei and nerves, kinesodic and æsthesodic, make up arcs for reflex actions of the most important kind, as breathing, swallowing, and (with gray matter in the pons) the state of vascular tonus. The respiratory centre (so-called) is in the nuclei of the pneumogastrics, the vaso-motor centre lies near the median line at the junction of the medulla and pons, and there are various subordinate foci or centres for important morbid actions, as the diabetic centres, the albuminuric centre, etc. Besides, the hypoglossal nuclei and nerves preside over the movements of the chief organ of speech.

I should add that the medulla oblongata has intimate though ill-understood connections with the cerebellum, by means of the restiform and olivary bodies.

As regards the localization of disease in the medulla, I shall have but little to say, for the reason that, in accordance with the terms of the course, I am bound to speak of only well-determined clinical forms whose lesions can be diagnosticated. Consequently, I will say nothing of systematic lesions in the bulbar æsthesodic system, nothing of bulbar lesions in diabetes mellitus, almost nothing of focal lesions of the organ.

a. Systematic lesions of the kinesodic tract are represented by only one typical symptom-group, viz.: the so-called labio-glosso-laryngeal paralysis, or bulbar paralysis. The lesion in this disease consist of granular degeneration of the ganglion cells of the nuclei of origin of the hypoglossus, spinal accessory, and of part of the facial nerves. The alteration is very like that present in progressive muscular atrophy (systematic lesions of the spinal cord, No. 5, Lecture III.), a molecular death, by degeneration, of ganglion cells.

The symptoms of typical labio-glosso-laryngeal paralysis are strictly motor, and consist in fibrillary contractions in and atrophic paralysis of the orbicularis oris, muscles of the tongue and throat, and some muscles of the larynx. Labial sounds are interfered with, saliva dribbles from the mouth, forcible blowing or whistling becomes impossible, the tongue plays heavily in the mouth as shown by thickness of speech and by difficult mastication, the voice becomes nasal through palatal paresis, and hoarseness, almost aphonia, is produced by palsy of the laryngeal muscles. At the same time, in extreme cases, the lower facial muscles become inert, the lower jaw hangs down, and an abundance of tenacious saliva runs from the patient's mouth; speech is quite unintelligible, and swallowing performed with extreme difficulty. At the close of life, if that be not cut short by the lodgment of food in the larynx, there are symptoms of injury to the pneumogastric nucleus, such as exceedingly rapid pulse and sudden stoppage of respiration.

In certain non-typical cases the lips, tongue and throat are affected in very various degrees; in others the pneumogastrics suffer early; and in others still the symptoms of progressive muscular atrophy, or of amyotrophic lateral sclerosis (Lecture II.) set in simultaneously or subsequently. I am disposed to believe that we may have many forms of "bulbar paralysis," and consequently would use the term as a generic one to embrace certain varieties, only one of which is now well known.

[It will be asked why I do not speak at length of descending degeneration through the medulla. This lesion is the continuation downward of a degenerative change in the motor tract beginning in the cerebral cortex, the internal capsule, the nuclei of the corpus striatum, or in the basis cerebri.

It usually affects only one anterior pyramid, and is continued downward into the cord, in its direct and crossed pyramidal columns. No symptoms indicate this alteration in the medulla, and we infer its existence from the diagnosis of descending degeneration in the spinal cord (Section 3, Lecture II.). I do not formally treat of this systematic bulbar lesion for the reason that it has no semeiology.]

b. With respect to focal lesions. A lesion involving one lateral half of the medulla will produce, first, symptoms of injury to the kinesodic and æsthesodic systems in that half of the medulla, the symptoms being on the same side as the lesion; and second, hemiplegia of motion (and sensation, if the injury be deep enough) in the opposite side of the body. Again, a superficial focal lesion involving the anterior (inferior) face of the medulla may produce symptoms closely resembling those of bulbar paralysis of the type labio-glosso-laryngeal paralysis; and perhaps the only way of reaching a correct diagnosis is by determining the presence of the degeneration-reaction in the paralyzed facial and lingual muscles, and by demonstrating weakness or positive palsy of the extremities.

An exceedingly atypical form of bulbar paralysis is the one caused by ischæmia of the medulla, owing to arrest of circulation in the vertebral and anterior spinal arteries. The symptoms are mixed motor and sensory (deglutition and articulation impaired, breathing of the Cheyne-Stokes type, rapid pulse, tottering gait or marked general paralysis) and death rapidly ensues.

LECTURE V.

SUMMARY:—LESIONS OF THE BASIS CEREBRI; PHYSIOLOGICAL ANATOMY OF THE PARTS INVOLVED. 1. LESIONS OF THE PONS VAROLII. 2. LESIONS OF THE CRURA CEREBRI. 3. LESIONS OF THE BASAL PARTS FORWARD OF THE CRURA; HEMIOPIA AND NEURO-RETINITIS.

GENTLEMEN:—Leaving the region of the medulla, we are brought to those numerous and important parts which constitute the encephalon. I purpose considering in some detail, in this and subsequent lectures, the localization of disease in the chief subdivisions of this mass, but in this lecture I can only enunciate some general pathological propositions relative to the encephalon as a whole, and study the lesions of one of its parts.

For our purpose I make the following subdivision of the encephalon—a semi-physiological classification:

1. The basis cerebri, including all the parts which lie upon the base of the skull, but more especially the pons Varolii, crura cerebri, their attached nerves, and the optic and olfactory apparatuses.
2. The great basal ganglia, *i.e.*, the thalamus opticus, nucleus caudatus, nucleus lenticularis, and the corpus quadrigeminum.
3. The white substance of the hemispheres, especially the internal capsule.
4. The cortex cerebri.
5. The cerebellum.

The general pathological propositions relative to these parts are as follows:

1. Lesions of the basis cerebri, especially if involving the pons and crura, give rise to the following symptoms: paralysis (often of crossed variety), anæsthesia in the face and limbs, impairment of equilibrium, changes within the eyes; no psychical symptoms.
2. Lesions of the great basal ganglia probably produce no symptoms unless by encroaching upon the internal capsule which passes near them. An exception may be the nucleus caudatus.
3. Lesions of the white centre of the hemispheres produce no symptoms when they do not involve the parts composing the internal capsule; if the anterior portion of this capsule be injured, we observe paralysis, if its posterior part, anæsthesia.

4. Lesions of the cortex cerebri produce, when located anteriorly, psychical symptoms; when located in the median regions, paralysis of an imperfect kind, and when situated posteriorly, no symptoms at all (sensory symptoms in animals).

5. Lesions of the cerebellum produce no symptoms except by involving adjacent parts containing important motor and sensory tracts; thus giving rise to irregular paralyses, changes in the optic apparatus, symptoms of irritation of the vagus nerve, etc.

6. Lesions in one lateral half of any part of the encephalon produce motor and sensory symptoms in the side of the body opposite to the lesion. When the lesion is in one-half of the basis cerebri some symptoms (direct symptoms) are found in the side of the face and head corresponding to the lesion, others in the opposite half of the body (crossed paralysis).

7. Lesions in the median line cause symptoms to appear in both sides of the body.

8. Any intracranial lesion which acts in such a way as to increase the intracranial pressure may produce (in addition to other symptoms) the condition known as choked disk, or neuro-retinitis.

With these preliminary general statements, I pass to the study of the first of the subdivisions—the basis cerebri:

PHYSIOLOGICAL ANATOMY.

To be very logical, the medulla oblongata, pons, and crura should be grouped together under this denomination, but for clinical purposes I leave the medulla with the spinal cord, and add to the basis cerebri the parts which lie in front of the crura, viz., the optic tracts and nerves, and the olfactory apparatus.

As heretofore, I assume that you are familiar with the descriptive anatomy of the parts; and what I wish you particularly to understand is the arrangement of sensory and motor tracts, of nerves, and of ganglia in the basis cerebri.

The projecting mass we call the pons is largely made up of numerous transverse nerve-fibers which connect it with the two halves of the cerebellum, and which are not, under our present knowledge, of special physiological or pathological importance. In the anterior (inferior) half of the pons, lying under those transverse fibers and, partly separated into bundles by them, we find the great motor or peduncular tract, the continuation upward of the anterior pyramids and of the central motor fasciculi of the medulla. This tract is easily seen with the naked eye, broken up into a number of smaller fasciculi by transverse fibres, and it can be traced upward a long distance. In the posterior (superior) region of the pons, under the floor of the fourth ventricle, near the median line, there is a lengthened mass of motor gray matter, whence arise the original fibres of the sixth, seventh, and motor root of the trigeminus nerves. Laterally, in the posterior part of the pons, are sensory tracts, some directly continuous with the brain above, others more particularly in relation with the sensory root of the trigeminus, which radiates to an immense extent up and down in the basis cerebri and below it.

In the crura cerebri we find substantially the same parts not covered over by transverse fibres. The anterior portion of the crura consists of the great motor or peduncular tract just issued above from the white matter and basal ganglia of the cerebrum. Posteriorly are sensory tracts and masses of gray matter in intimate relation with the thalamus opticus and corpus quadrigeminum. The final (upper) extremity of the elongated mass of gray matter giving rise to motor nerves is found here, posteriorly, just beneath the aqueduct of Sylvius, in the shape of the common nucleus of the third and fourth nerves. Let us finally bear in mind that the sixth, seventh, and fifth nerves are intimately connected with the pons Varolii; the third and fourth nerves, and the optic tracts are associated with the crura.

The posterior portions of the crura and pons constitute the greater part of what Meynert designates the *tegmentum cruris cerebri*, while their anterior parts contribute to form his *basis cruris cerebri*.

All these parts lie in the middle fossæ of the cranium, the chiasm of the optic nerve occupying their forward extremity. In the anterior fossæ we find only the olfactory commissures and ganglia, together with the under surface of the frontal lobes of the cerebrum.

As regards the physiology of these parts, the mesencephalon of some authors, we may sum it up as follows:

The anterior part of the crura, pons (apart from the superficial transverse fibres), and medulla oblongata, contains the chief motor tract connecting the superior centres with the spinal cord. This motor tract is made up of nerve fibres, which convey excitations chiefly in a centrifugal direction; many of them, derived from the motor regions of the cortex, constitute the anterior portion of the internal capsule, and then enter the crura, traverse the pons, go to make up the anterior pyramids of the medulla, partially and irregularly decussate at the pyramidal decussation, and finally are found in the postero-lateral and anterior columns of the spinal cord. This most important bundle of nerves may be designated as the *direct cerebral motor tract.* Another portion of the anterior region of the mesencephalon is likewise motor in function, and is made up of bundles of fibres derived from the nucleus caudatus and nucleus lenticularis. These bundles can be traced downward into the middle regions of the medulla, but their connection with the spinal cord is yet uncertain. The posterior region of the pons and crura, composed largely of gray matter, is partly sensory and partly motor. It is motor only in so far as it includes the nuclei of origin of the upper cranial nerves, the seventh, sixth, motor root of fifth, the fourth and third. You will remember that the nuclei of these nerves are all to be found near the median line, underneath the floor of the fourth ventricle or its continuation, the aqueduct of Sylvius. The sensory parts of the mesencephalon embrace the regions lying external to these nerves, giving origin to the eighth and fifth pairs of nerves; the latter presenting an enormous expansion in its origin; its upper roots extending as high as the region of the corpus quadrigeminum, the lowest probably as far down as the upper part of the spinal cord.

The posterior regions of the mesencephalon, the *tegmentum cruris,* are the seat of the reflex actions of the most important character, and they probably serve also for the elaboration, if not perception, of sensory impressions from the periphery. At the upper part of the medulla and the lower part of the pons in this region is a mass of gray matter which controls the vaso-motor phenomena throughout the body. It is highly probable that some severe convulsive manifestations, such as epileptic and tetanic seizures, are due to morbid processes in the posterior part of the pons and crura.

Finally, with respect to the physiology of the optic apparatus lying at the base of the brain, I will only say that I am disposed to accept the doctrine of semi-decussation of the optic tracts in the chiasm, and shall use this hypothesis in explanation of symptoms.

SYMPTOMS OF LESIONS OF THE BASIS CEREBRI.

Before taking up systematically the study of the semeiology of basal lesions, allow me to fully discuss one of the most frequent symptoms of all these lesions, viz., crossed paralysis. I wish to study crossed paralysis in general, previous to speaking of it under each heading of the remainder of this lecture, partly to impress you with its importance, and partly to avoid future digressions and repetitions. By crossed paralysis (*paralysie alterne* of the French) is meant a form of paralysis in which the symptoms immediately caused by a basal lesion are on one side of the face or head and on the same side as the lesion, while the bodily symptoms are on the opposite side of the median line, viz., on the side opposite the lesion. As thus enunciated, in principle, it is at once apparent that the phenomena of crossed paralysis may, according to the seat of a lesion at the base of the brain, involve, on the one hand, any one of the cranial nerves, and on the other the limbs of the opposite side. This conception is verified by clinical and post-mortem experience, cases of crossed paralysis of every possible variety being on record. The most striking and best known, however, are those presenting what I may be allowed to term the third nerve and body type, the trigeminus and body type, and the seventh nerve and body type. It is to the late Prof. Romberg, of Berlin, that we owe the exact definition and conception of the principle of crossed paralysis, while Prof. Gubler, of Paris, first made a thorough study of the seventh nerve and body type.

1. LESIONS OF THE PONS VAROLII.

Diffused lesions of the pons produce, when fully developed, partial anæsthesia and paresis on both sides of the face, generalized paralysis below the neck, with or without anæsthesia, the latter symptom appearing only if the deeper, posterior, portions of the organ are involved. We also observe inability to maintain equilibrium, without ataxia, sometimes convulsions, or contracted pupils, or neuro-retinitis. If the lesion advance forward beyond the pons, new symptoms such as will be described further on are superadded.

Localized lesions of the pons may occupy on either side of the median line one of four locations.

a. In the anterior portions of the pons forward of an imaginary transverse line passing through the origin of the trigemini. As shown by Gubler, this lesion does not produce crossed paralysis of the seventh nerve and body type, but both the face and body are paralyzed on the same side, *i.e.*, on the side opposite the lesion, just as in cerebral lesions strictly speaking. A point for differential diagnosis is that when the cerebrum is injured, there is almost invariably conjugate deviation of the head and eyes toward the affected hemisphere; in pons lesions nothing of the kind occurs.

b. A lesion placed so as to injure the anterior (inferior) region of the pons on one side, back of the imaginary line above mentioned, will cause typical crossed paralysis, *i.e.*, the facial nerve will be paralyzed on the same side as the lesion, and the extremities on the opposite side. For example, if the right face and the left arm and leg be palsied in a patient, we recognize crossed paralysis of the seventh nerve and body type, and may diagnosticate with positiveness a lesion placed as above described. There is, perhaps, no more positive example of constant relation of lesion to symptoms in the whole of nervous pathology.

In *a* and *b*, if the trunk of the trigeminus be involved in the disease, the face will be more or less anæsthetic and neuralgic on the same side as the facial palsy and the lesion. From a study of cases of these two kinds, Gubler drew the anatomical conclusion that the paths which connect the cerebrum with the nuclei of the seventh nerve decussate at about the middle of the pons, *i.e.*, some distance above the nuclei of these nerves. A useful diagram for studying the principle of crossed paralysis, and of this type in particular, you will find in Dr. Hammond's Treatise on Diseases of the Nervous System, ed. 1876, p. 99.

c. Lesions occupying the posterior region of the pons above its middle. Besides paralysis of the face and body on the side opposite the lesion, we are likely to have anæsthesia of the paralyzed parts, even amounting to hemi-anæsthesia (without involvement of the special senses). Other symptoms often produced are epileptic convulsions, impairment of sight from neuroretinitis, and various forms of paralysis of ocular muscles.

d. Lesions in the posterior part of the pons, below the imaginary transverse line through its equator, may likewise, if extensive, without crossing the median line, cause hemi-anæsthesia of common sensory nerves, but will also produce crossed palsy, of face on same side as the lesion, of the body on the opposi side. As in *c*, we may have neuro-retinitis, epileptic seizure with, besides, palsy of the sixth nerve, and bulbar symptoms the lesion involve the medulla.

e. A lesion situated very laterally in the pons, or so placed a to irritate the lateral peduncles of the cerebellum, will (as show by a number of cases) give rise to rotatory movements of th patient around the long axis of his body toward the side of th lesion. Of course this symptom appears with others, which ar more or less in accord with the above symptom-groups.

2. LESIONS OF THE CRURA CEREBRI.

a. Lesions of the crus proper on one side of the median line. The symptoms of such a lesion are exceedingly definite, and might even be designated pathognomonic. The third nerve, its trunk or origin, is involved in the disease or compressed, as well as the great motor tract which, lower down, is to decussate. Consequently we observe a crossed paralysis of the third nerve and body type; *i. e.*, if the right motor oculi, the left lower face and the left extremities be paralyzed in a patient, we may feel sure that he has a lesion under or in the right crus cerebri. Prof. Rosenthal, of Vienna, states that in such cases the electro-muscular contractility (to faradism) is reduced in the paralyzed limbs, contrary to what obtains in hemispheric lesions. You will remember that the optic tract curves around the crus on either side, and it at times happens that symptoms characteristic of injury to one optic tract present themselves. These symptoms will be considered later.

b. Lesions deeply placed in the crus, on one side of the median line. In addition to the above described crossed paralysis, we shall probably observe paralysis of the lower part of the face on the side opposite the lesion and hemi-anæsthesia of common sensation only on the side opposite the lesion. There may be convulsions, choreiform spasm, hemiopia, or neuro-retinitis.

c. Lesions placed in the median line, or involving both crura more or less. The symptoms will be, in case of lesion situated anteriorly (inferiorly), wholly motor, viz.: paralysis of both third nerves; the lower half of both sides of the face and of both sides of the body below the neck. In case of lesion involving the tegmentum cruris, marked disorders of sensibility, neuro-retinitis, and convulsions will occur.

Extension of the disease backward to the pons will be characterized by symptoms (detailed in first paragraph) referable to the trigeminus and seventh nerves.

It has been stated that the bladder is paralyzed in severe lesions of the crura, but this lacks confirmation.

It might not be ill to add that patients having lesions in the locations defined above, do not present, strictly speaking, cerebral symptoms. They preserve their intellect, are not aphasic, and the special senses (except the sense of sight) are not involved.

3. LESIONS SITUATED AT THE BASE OF THE BRAIN ANTERIORLY TO THE CRURA.

These are rare, but give rise, when considerable, to noteworthy symptoms.

a. Lesions involving one of the optic tracts posterior to the chiasm. A small lesion in this location will produce hemiopia, a symptom of such importance and scientific interest as to require special mention. If the lesion extend, it may involve the chiasm of the optic nerves, or the tract of the opposite side, or the crura cerebri, thus either destroying the special symptom, hemiopia, or adding to it the signs of crossed paralysis of third nerve and body type.

By hemiopia is meant a condition of the organs of vision, such that only one-half (vertically divided) of objects are seen by the patient. In other words, the fields of vision of the two eyes are darkened in one of their halves. Usually the vertical line of division between the lighted and darkened half-fields is exactly in the centre of vision. The hemiopia affects the two eyes always, and is distributed differently in each eye under the determining influence of the exact location of lesions about the optic tracts and chiasm. The immediate cause of hemiopia is interruption in the centripetal conducting power of certain bundles of nerve-fibres in the optic nerves and tracts; hence it has been found necessary to frame a theory of the course of fibres in the optic apparatus. The hypothesis of Wollaston, that of semi-decussation, is the one which is generally adopted to-day, and I append a diagram illustrating it, and the positions of lesions producing the varieties of hemiopia. A satisfactory anatomical demonstration of the truth of the theory of semi-decussation has not yet been given, and some few facts and authorities are opposed to it, but I think that I can assure you that in the present state of science it is the one which best serves the purposes of the physiologist and the clinical observer.

According to this hypothesis nerve-fibres derived from the deep origins of one (the left) optic tract, combine to form that bundle, and extend undivided as far as the chiasm. As shown in Fig. 6, at the chiasm some of the fibres of the tract (the external group possibly) pass directly into the external portions of the left optic nerve, and are distributed to the external (temporal) half of the left retina. The remaining nerve-fibres of the left optic tract cross the median line in the middle of the chiasm, decussating with the similar fibres derived from the right tract, then enter the right optic nerve, and are distributed to the inner (nasal) half of the right eye. Thus it is seen that the nasal half of the right eye and the temporal half of the left eye are anatomically and physiologically homologous.

The same explanation applies to the course of fibres which form the right optic tract. Besides these main bundles of fibres, a few filaments are supposed to connect the two retinæ, passing through the optic nerves and the anterior border of the chiasm without decussation, while others connect the two sets of tubercula quadrigemina by way of the optic tracts and the posterior border of the chiasm. These commissural fibres may, however, be left out of consideration in the study of hemiopia.

If now we bear in mind this distribution of fibres in the optic apparatus, we can, by the aid of Fig. 6, demonstrate the mechanism of each of the four forms of hemiopia, viz.: right and left lateral hemiopia (shaded parts A and B), bi-temporal and nasal hemiopia.

Lateral hemiopia, *i. e.*, that form of hemiopia in which the homologous parts of the two eyes (temporal half of the left retina and the nasal half of the right) lose their function, is invariably produced by a lesion destroying one optic tract—the left in the diagram. *Vice versa*, destruction of any part of the right optic tract would cause blindness in the temporal half of the right retina and in the nasal half of the left. Inasmuch as all visual rays cross in the eyes, owing to refraction in the lens, the hemiopia, *i. e.*, the obscuration of the half-fields of vision, is on the opposite side from the blind-half retinæ. For example, in the first instance described above and illustrated in the diagram, the hemiopia is of the form designated right lateral (or homonymous) hemiopia, and is produced by a lesion involving the left tractus opticus.

Binocular temporal hemiopia can be caused only by a lesion placed (as 1 in Fig. 6) in front of the chiasm, cutting off the fibres which supply the two nasal fields.

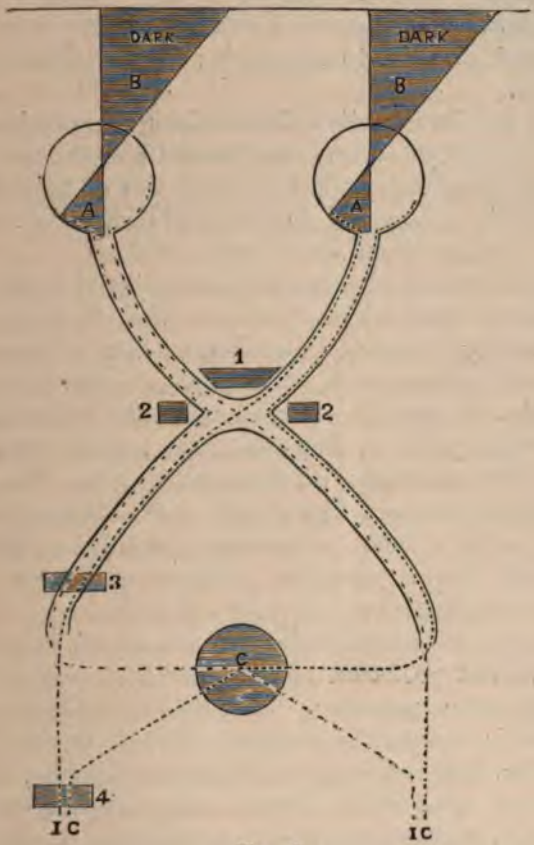

Fig. 6.

Diagram explicative of hemiopia. The shaded intra- and extra-ocular parts, A and B, indicate the obscuration in right lateral (or homonymous) hemiopia, as caused by lesion 3, so placed as to destroy one optic tract. In that tract are two sets of nerve fibres, one represented by a dotted line supplying the nasal half of right retina, the other fibres by a broken line supplying the outer or temporal half of the left eye. As visual lines cross in the eye the obscuration of the half-fields is the opposite. Lesion No. 1, anterior to chiasm, produces blindness of inner half of each retina, and consequently bi-temporal hemiopia. Lesions No. 2, pressing upon the sides of the chiasm, injure fibres supplying the temporal half of each retina, and cause bi-nasal hemiopia. C., corpus quadrigeminum, in which Prof. Charcot believes a second partial decussation takes place. I.C. Internal capsule containing, on Charcot's hypothesis, all the fibres coming from the eye of the opposite side. 4. Lesion of internal capsule injuring all the fibres connected with the right retina, and causing amblyopia of the right eye.

Binocular nasal hemiopia is produced by a double lesion compressing or destroying the outer parts of the chiasm (2 in Fig. 6), which are imbedded fibres supplying the temporal halves of both retinæ. Prof. Knapp, of New York, has placed a remarkable case of this kind on record, in which the lesion consisted of thickened and enlarged internal carotid arteries. I need hardly add that lesions involving one optic nerve in front of the chiasm cannot give rise to hemiopia; they produce monocular loss of vision.

It should not be forgotten that lesions lying in front of the crura and behind the chiasm may press upon the motor nerves of the eye as they traverse the middle fossæ of the cranium on their way to the sphenoidal fissure, thus producing a variety of paralytic symptoms about one or both eyes.

b. Lesions in the anterior cranial fossa, anterior to the chiasma. When unilateral, such a lesion involves the olfactory tract and ganglion, thereby producing anosmia, or loss of smell, on the same side as the lesion, with or without subjective odors.

If the lesion be large, it may act upon the nucleus caudatus or other motor parts of the encephalon, and cause common hemiplegia (face and limbs) on the opposite side. Thus we may have a last form of crossed paralysis, of the olfactory nerve and body type. With a lesion involving both sides of the median line, complete double anosmia, with or without generalized paresis of the extremities, would be met with.

There is one more symptom common to all lesions of the basis cerebri, but produced also at times by any intracranial disease which causes pressure. I mean neuro-retinitis or choked disks. This is always (?) bilateral, though it may be more marked in one eye, and is esteemed one of the most important signs of gross encephalic disease, especially of tumors. In neuro-retinitis, ophthalmoscopic examination shows that the optic nerves are swollen, and they may project considerably (measurably) above the level of the surrounding retina; the margin of the disk is obscured or wholly lost, and no line of demarcation can be made out between the nerve and the retina. The blood-vessels present striking anomalies, the arteries being relatively small, the veins positively enlarged and tortuous; there are often small hemorrhages in the retina, round about the disk. Strange to say, very good sight may coexist with this lesion. This condition of choked disks may last a number of weeks (much longer in cases of tumor of the brain), and then subside, giving place to the appearances of atrophy of the optic nerves, viz., an unnatural whiteness or bluish whiteness of the disk, smallness of the retinal vessels, and unusual sharpness of the outline of the disk, with impaired vision.

LECTURE VI.

SUMMARY:—LESIONS OF THE GREAT GANGLIA AT THE BASE OF THE BRAIN, AND OF THE WHITE SUBSTANCE OF THE HEMISPHERES.—SKETCH OF THE PHYSIOLOGICAL ANATOMY OF THESE PARTS: LESIONS OF THE GREAT BASAL GANGLIA OF THE INTERNAL CAPSULE, AND OF THE REMAINDER OF THE WHITE SUBSTANCE.—LESION OF THE CEREBELLUM.

GENTLEMEN:—The great ganglia at the base of the brain are from before backward, the nucleus caudatus and the nucleus lenticularis of the corpus striatum (the intra- and extra-ventricular portions of the corpus striatum, according to English and American books), the thalamus opticus, and the corpus quadrigeminum (or tubercula quadrigemina). These masses of gray matter are of course double, *i.e.*, symmetrically arranged on either side of the median line. Their relations to adjacent portions of the brain are of great importance to us, and worthy of a somewhat detailed study.

In very general terms, it may be said that all of these bodies have at least a duplex connection, one superior, with the cortex of the brain, the other inferior, with the various parts which make up the mesencephalon. More particularly, the nucleus caudatus (intra-ventricular nucleus of the corpus striatum) sends bundles of fibres downward into the crura, and the same is true of the externally-placed nucleus lenticularis. So we say that both parts of the corpus striatum are intimately connected with the basis cruris cerebri.

On the other hand, the thalamus and corpus quadrigeminum are intimately united with the nucleus of the tegmentum, and send bundles of fibres into and through it, in the posterior or sensory system of the mesencephalon. Physiologically, we may look upon the basis cruris and its superadded ganglia as motor in function, and upon the tegmentum cruris as sensory and as the seat of performance of important and complex reflexes.

As regards the other connections of these bodies, they are probably connected with their homologues across the median line, and superiorly with various parts of the cortex. There would seem to be, judging from a series of cases of cerebral atrophy, a bundle of nerve fibres connecting the nucleus caudatus of one side with the opposite half of the cerebellum by way of the processus cerebelli ad cerebrum. At any rate, great atrophy of the right hemisphere (for example) and corpus striatum is usually accompanied by atrophy of the left hemisphere of the cerebellum. I need hardly remind you of the intimate union between the optic tracts and the corpus quadrigeminum and the external portion of the thalamus.

The white centre of the hemispheres is made up of many separable fasciculi, the physiology of many of which is as yet obscure. In the first place, we can demonstrate in it commissural bundles running in various directions: transversely, connecting various portions of the cortex of both hemispheres by way of the corpus callosum and the so-called commissures; others extend longitudinally in one hemisphere, connecting the convolutions of one lobe with those of another; yet others simply bind together adjacent convolutions. Secondly, there are heavy masses of fibres extending from the basal ganglia to the convolutions of the hemispheres, constituting a great part of what is known as the corona radiata. Thirdly, and most important in pathology to-day, is a bundle of white substance in each hemi-

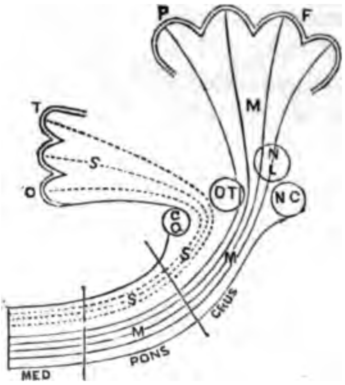

FIG. 7.

Diagram of course of sensory and motor tracts in the mesencephalon and hemispheres. S, sensory tract in posterior region of mesencephalon, extending to O and T, occipital and temporal lobes of hemispheres; M, motor tract in basis cruris, extending to P and F, parietal and (part of) frontal lobes of hemispheres; C Q. corpus quadrigeminum; O T, optic thalamus; N L, nucleus lenticularis; N C, nucleus caudatus.

sphere, which directly unites the cortex of the brain with the crura, pons, and spinal cord—the so-called internal capsule. This fasciculus appears (upon anatomical and physiological evidence) to be the continuation of the sensory and motor tracts which we have studied in the basis cerebri. The extension of the sensory tract upward from the crura into the internal capsule is not so clear as is the continuity of the motor tract anteriorly. This, denominated by some the peduncular tract, and designated in Lect. V. as the *direct cerebral motor tract*, can be traced by dissections, by physiological experiments, by embryology, and lastly by the help of pathological processes (descending degeneration) from certain convolutions of the cortex cerebri into the anterior half or two-thirds of the internal capsule, into the crus cerebri, the pons, the medulla, and (in accordance with the law of decussation—*vide* Lect. IV.), into both halves of the spinal cord. The composition and extension of the internal capsule is rudely represented in diagram by Fig. 7. Its exact position in the hemisphere and its relations to other parts of the encephalon are well shown in Fig. 8, modified from a cut in Prof. Charcot's

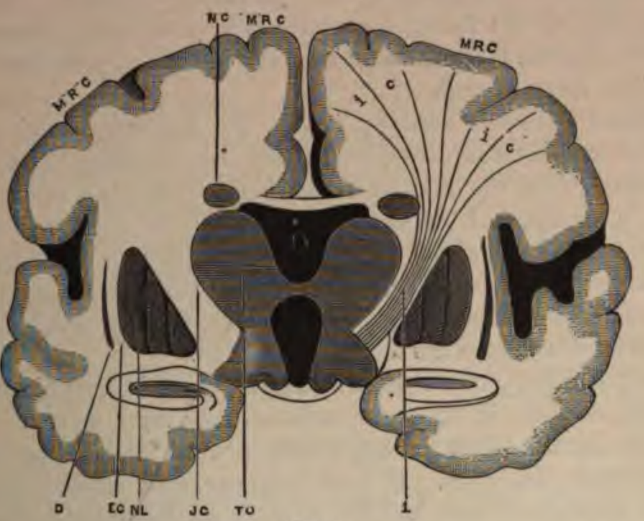

FIG. 8.

Modified from Charcot's diagram, to show position, relation, and distribution of the internal capsule as seen in a vertical transverse section of the brain on a level with the greatest development of T O, thalamus opticus. I C, location of internal capsule; N L, nucleus lenticularis; E C, external capsule; D, claustrum; N C, nucleus caudatus; M R C, motor regions of cortex cerebri; 1, fibres representing the radiation of the internal capsule vertically to the motor region of cortex.

Lectures on Localization in Cerebral Diseases. This represents a transverse vertical section of the hemispheres made through the middle of the optic thalamus. The internal capsule is seen to lie between the external border of the thalamus and the nucleus lenticularis of the corpus striatum. In this location the bundle is much compressed, and seems to have little or no connection with the gray bodies just named; it expands in every direction after passing them, and its anterior fibres are ultimately distributed to the middle regions of the cortex cerebri—motor district—embracing the third frontal, the posterior extremities of the first and second frontal, the ascending frontal convolutions, also the ascending parietal and first parietal convolutions; also, lastly, to the continuation upon the inner surface of the hemisphere of the ascending frontal and parietal convolutions which make up the paracentral lobule. The posterior third of the internal capsule is very probably distributed to the occipital, temporal and second parietal convolutions—the supposed sensory gyri.

Besides common sensory tracts, the posterior part of the internal capsule contains fibres which are directly or indirectly connected with the special sense organs, viz., the optic, olfactory, gustatory, and acoustic. This is shown by the fact that total hemi-anæsthesia in man results from lesions involving the internal capsule; from experiments upon animals, whereby certain occipital or temporal convolutions being removed, blindness or deafness is caused on the opposite side.

The physiology of the great ganglia, commissural fibres, and internal capsule is far from satisfactorily marked out. Many glaring contradictions are apparently proved by the experiments of different observers. It would appear well settled that the corpus quadrigeminum and the corpora geniculata are a part of the optic apparatus; that the nucleus caudatus and the nucleus lenticularis have motor functions of some sort. The greatest uncertainty exists, I think, concerning the attributes of the thalami optici.

On the other hand, we now know pretty positively that most of the sensory paths for the body pass in the posterior part of the internal capsule, laceration of this part in animals being invariably followed by hemi-anæsthesia on the opposite side. That the thalamus has anything to do with the perception of sensations is rendered doubtful by the occurrence of cases in which lesions, strictly limited (*i. e.*, not pressing on adjacent parts) to the thalamus have produced no special symptoms. The same objection (pathological) can be urged against the view that the nucleus lenticularis has important motor functions. That the anterior part (two-thirds?) of the capsule consists of motor fibres is likewise quite well established by experiments upon animals and by the study of pathological cases.* What the functions of the various commissural fibres may be, is, at the present time, only a matter of speculation more or less logically constructed.

1. LESIONS OF THE BASAL GANGLIA.

The generally received statements that lesions of these bodies produce definite symptoms—lesions of the nucleus lenticularis and nucleus caudatus paralysis, and lesions of the thalamus opticus, anæsthesia—are, I think, very questionable. From the evidence now before us it seems doubtful if lesions of the nucleus lenticularis, and of the thalamus, produce any symptoms except by exerting pressure upon the internal capsule lying near by. At any rate, cases of destruction of large parts of these bodies without symptoms are on record. As regards the nucleus caudatus, it is possible that its destruction is followed by hemiplegia and secondary degeneration; but on the other hand, it must be admitted that almost all lesions of this body are so placed, and of such a nature, as to cause pressure upon the motor portion of the internal capsule. This is especially true of hemorrhage, as shown by Prof. Charcot in his recent lectures on the brain.

2. LESIONS OF THE INTERNAL CAPSULE.

These, on the other hand, produce, as shown by the recent study of pathological evidence, and by experiments upon animals, constant symptoms. If the anterior half or two-thirds of the capsule be injured, we have hemiplegia on the opposite side of the body; more or less perfect hemiplegia according to the exact seat and size of the lesion. It will be readily understood that lesions (especially hemorrhage and tumors) of parts adjacent to the capsule, such as the lenticular and caudate nuclei, the white centre of the frontal lobe, etc., may by pressure bring about a similar result. Besides paralysis, descending secondary degeneration is an inevitable result of lesions of the internal capsule. This lesion can be traced downward through the whole length of the direct cerebral motor tract so frequently referred to before, to the lower end of the spinal cord. Lesions of the nucleus caudatus are also said to produce the same results, but recent researches (Flechsig) throw doubt on this.

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* Since these lectures were delivered, a brilliant and final proof of the motor functions of the anterior part of the internal capsule, and of the continuity of the motor convolutions and the direct cerebral motor tract has been advanced by the French experimenters, Franck and Pitrès (*Le Progès Médical*, Jan. 19, 1878). These physiologists found that by faradizing those parts of the white centre of the hemisphere which lie underneath the so-called cortical motor centres, they were able to produce definite movements in parts of the body on the opposite side. This tract of white matter, constituting the anterior portion of the internal capsule, contains physiologically distinct fasciculi which are connected on the one hand with the motor districts of the cortex, and on the other hand with peripheral parts of the body across the median line.

If the posterior part of the internal capsule be injured directly, or indirectly by the pressure of lesions in adjoining regions, there is produced anæsthesia, more or less complete, on the opposite side, usually with only slight paralysis. If the lesion be considerable the anæsthesia is absolute; *i.e.*, the special senses and common sensory nerves lose their function, or more properly speaking, impressions coming through these cannot reach the perceptive centers. Hemiopia is never (?) thus produced, and Charcot explains this by supposing a second semi-decussation to take place in some part between the chiasm and the internal capsule; probably in the corpus quadrigeminum. A reference to Fig. 6, Lect. V., will, I trust, make this hypothesis plain.

Yet a third symptom results from lesions of the internal capsule, viz., choreiform movements following hemiplegia and hemi-anæsthesia. These movements vary in degree and type from true athetosis to ataxia, from chorea to tremor, and constitute an interesting symptom-group, well worthy of further study. In my own cases of post-hemiplegic chorea, hemi-anæsthesis of slight degree was present, and in one case lateral hemiopia.

Extensive lesions of the central parts of the hemispheres may produce, besides the specific signs named above, a number of other symptoms. Thus, suddenly produced lesions (hemorrhage, softening) will nearly always cause conjugate deviation of the eyes and head. The patient, though insensible, turns his eyes and head constantly to one side, toward the injured hemisphere, and away from the paralyzed side. In spite of a recent attempt to impeach the value of this symptom of hemispheric injury, I am disposed to attach value to it. Great increase in the bodily temperature also follows large injuries.

Other lesions, such as cause pressure, and slowly grow to a great size (tumors), cause, in addition to the specific signs dependent upon their exact location, the change within the eye which we call neuro-retinitis, (*vide* Lect. V.). They will also usually produce convulsions.

As regards the remainder of the white substance, such as the central regions of the frontal, occipital, and temporal lobes, modern critical study of recorded cases would seem to indicate that lesions involving these parts in such a way as not to press upon the internal capsule and nucleus caudatus, do not give rise to any symptoms; *e.g.*, an immense abcess may occupy the temporal and occipital lobes, or the anterior part of the frontal lobe, without causing paralysis or anæsthesia. Anatomy and experimentation, however, seem to indicate that lesions of the occipital and temporal lobes should give rise to sensory symptoms; and a more careful study of cases of disease in these parts is just now a desideratum.

We do not know any more relative to lesion of the great commissural bundles which unite the two hemispheres and different parts of one hemisphere. The cases of congenital absence of the corpus callosum on record do not teach anything definite.

3. LESIONS OF THE CEREBELLUM.

I add a few words relative to another *terra incognita* in the brain, the cerebellum. Its situation is known to all of you, but there are a few points in its anatomy to which I would specially invite your attention. In the first place, this great mass of nervous matter is closely bound down by a strong fibrous covering (a bony septum in some animals), the tentorium cerebelli. This fold of dura mater probably serves important purposes in health, but in case of disease in the cerebellum it causes pressure-effects to be transmitted chiefly forward and downward. This is important to bear in mind when studying the effects of cerebellar lesions. Second, the cerebellum is remarkable for its numerous connections with other parts of the nervous system. Fibres connect each of its hemispheres with the nucleus caudatus and cerebral hemisphere of the opposite side, by means of the crura cerebelli ad cerebrum. Other fibres, forming heavy bundles, make up the crura ad pontem or lateral peduncles of the organ; extending deeply into the white and gray substances of the mesencephalon. It is probable that each half of the cerebellum is thus connected with the opposite half of the pons; possibly some fibres are strictly commissural, *i.e.*, unite the two hemispheres of the organ after passing over the pons. Lastly, the cerebellum is connected with the medulla and spinal cord. It forms, by means of the crura cerebelli ad medullam, close connections with the olivary bodies, and with the external portion of the lateral columns (near the extremity of the posterior horns) in the spinal cord. It has been claimed that some cranial nerves (third, fourth, and acoustic) have been traced into the cerebellum, but the evidence on this point is unsatisfactory. Third, the cerebellum overlies highly important organs, and this proximity serves to explain much of the semeiology of its lesions. In front of it is the corpus quadrigeminum and the tegmentum cruris, with its contained vaso-motor (and convulsive?) centre; beneath it the medulla oblongata, with its floor and such vital nerves as the pneumogastric and the spinal accessory.

The physiology of the cerebellum is at the present day quite unknown. That it serves for purposes of co-ordination in a direct and positive manner is disproved by experimentation and pathology; that it is a centre for the movements of the eyeballs (Ferrier) is equally doubtful; and so is the view that it is the seat of psychical attributes of an emotional character. Mitchell's hypothesis, that it is a store-house of nerve force for use in emergencies, is plausible but unproven. The mechanism and purpose of the cerebellar connections is likewise not understood.

As regards the diagnosis of lesions of the cerebellum, I must admit that, in the very numerous symptoms produced by them, I do not know of one that is characteristic. In other words, lesions strictly limited to the substance of the cerebellum produce no definite symptoms: and on the other hand, the symptoms which we observe in cerebellar diseases are the result of pressure-effects upon adjacent parts. Thus, the affections of sight so common in cerebellar lesions are caused by pressure upon the corpus quadrigeminum or the corpora geniculata, and also upon the origins of the third, fourth and sixth nerves. The nausea, vomiting, and sudden death may be explained by irritation and paralysis of the nuclei of the pneumogastric nerves in the floor of the fourth ventricle ; convulsions, by pressure upon the tegmentum cruris ; the imperfect hemiplegia or general paralysis, by a similar action upon the motor regions of the mesencephalon. The diagnosis (assisted by predominance of pain in the occipital region) must be made chiefly by exclusion. A symptom of great importance when present is titubation. This has been termed cerebellar ataxia, but as a descriptive term titubation is better. The patient walks with his feet separated, his body bent a little forward and swaying, his hands and arms in use to preserve his equilibrium. There is no true ataxic jerking, no want of harmony between antagonistic groups of muscles, no choreic movements, no tremor.*

LECTURE VII.

SUMMARY :—ANATOMY AND LESIONS OF THE CORTEX OF THE BRAIN.—THE CHIEF CORTICAL MOTOR CENTRES, AND BROCA'S SPEECH-CENTRE.—LOCALIZED LESIONS OF THE CORTEX CEREBRI ; DIFFUSED LESIONS OF THE SAME.

GENTLEMEN :—The cerebral cortex is an immense, spread-out ganglion, whose functions are not yet fully or exactly known. Like all ganglionic masses, it is composed of ganglion-cells, nerve-fibres, blood-vessels, and neuroglia. Its ganglion-cells are generally pyramidal in shape the apex of the pyramids being turned outward or peripherally. They vary very much in size and in precise shape, the largest occurring in convolutions of the median parts of the hemispheres.

This gray cortical layer becoming folded through the process of growth, ultimately presents irregular swellings and depressions of its surface. The swellings are called convolutions or gyri; the depressions, fissures or sulci. Some of the sulci are very deep, and receive special names. It should not be forgotten that the bottom of every sulcus is formed by the same ganglionic gray matter as the prominent parts of gyri.

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* After the delivery of this lecture, Prof. H. Nothnagel of Jena, published (in *Berliner klinische Wochenschrift*, 1878, No. 15) the results of his analysis of more than two hundred and fifty cases of cerebellar disease. His conclusions, I am happy to say, are substantially equivalent to what has been said above. However, Prof. N. is disposed to admit one symptom—cerebellar ataxia—as characteristic of injury to the cerebellum or more properly, to one of its smaller parts, the superior vermiform process. N. says that by cerebellar ataxia we are to understand a perversion of equilibrium closely resembling that observed in alcoholic intoxication; the patient titubates, stands with feet wide apart ; if he be barefooted the toes are seen in active motion : and in walking the body sways a good deal, the foot is brought down with ball or with heel first irregularly ; closing the eyes sometimes makes standing and walking worse, sometimes not. In the recumbent position there is no ataxia. In the large majority of cases the upper extremities remain free from inco-ordination.

These gyri are so grouped and separated by large sulci, that we are now enabled to make a successful topographical study of the apparantly confused mass of convolutions; and in my brief description of the cortex cerebri I shall almost follow Ecker's classification of its parts. Thus in each hemisphere we have four lobes or groups of gyri, viz., the frontal, parietal, temporal, and occipital lobes. Separating these lobes are three large and constant fissures: the fissure of Sylvius, between the frontal and temporal lobes; the fissure of Rolando (or central f.), separating the frontal and parietal lobes; and the occipito-parietal fissure in the inner face of the hemisphere, limiting the occipital and parietal lobes.

Besides, we recognize four lobules, viz., lobulus centralis (island of Reil at the bottom of the fissure of Sylvius), lobulus paracentralis, lobulus cuneus, and lobulus quadratus, on the inner surface of the hemisphere. The paracentral lobule is made up almost wholly of the upper (inner) ends of the ascending frontal and ascending frontal convolutions as they dip into the great longitudinal fissure.

For a full account of these parts I would refer you to Ecker's monograph on the cerebral convolutions, to Ferrier's work on the functions of the brain, and to the latest edition of "Dalton's Physiology." A few of the convolutions in these lobes and lobules are of importance in the study of localization, and I must briefly describe them.

First, the third and ascending frontal convolutions. The former of these (f. 3. Fig. 9) constitutes the lower tier of gyri in the external aspect of the frontal lobes, and forms the antero-superior lip of the fissure of Sylvius. Its posterior part and its continuation into the island of Reil certainly have a very close connection with the function of written and spoken speech. The ascending frontal gyrus (A, Fig. 9) forms the posterior limit of the frontal lobe, and lies against the fissure of Rolando. Ecker calls it the anterior central convolution, but in common with Prof. Charcot and his pupils, I prefer the former designation.

Immediately behind the fissure of Rolando, extending almost vertically, is the ascending parietal gyrus, or the posterior central convolution of Ecker. These two gyri, the ascending frontal and parietal, are intimately connected with movements of the face, arm, and leg; so-called centres for the face existing in the lower ascending frontal, centres for the upper extremities being found in its middle portions and in the ascending parietal gyrus; while the centres for the movements of the lower limbs are in the upper (inner) extremities of both these gyri, and in the next parietal convolution near the median fissure, viz., the superior parietal (p. 1, Fig. 9).

The last gyrus of clinical importance is the next below the inferior parietal and its extension toward the occipital lobe, the angular gyrus, so-called (P2 Fig. 9). Some recent clinical and

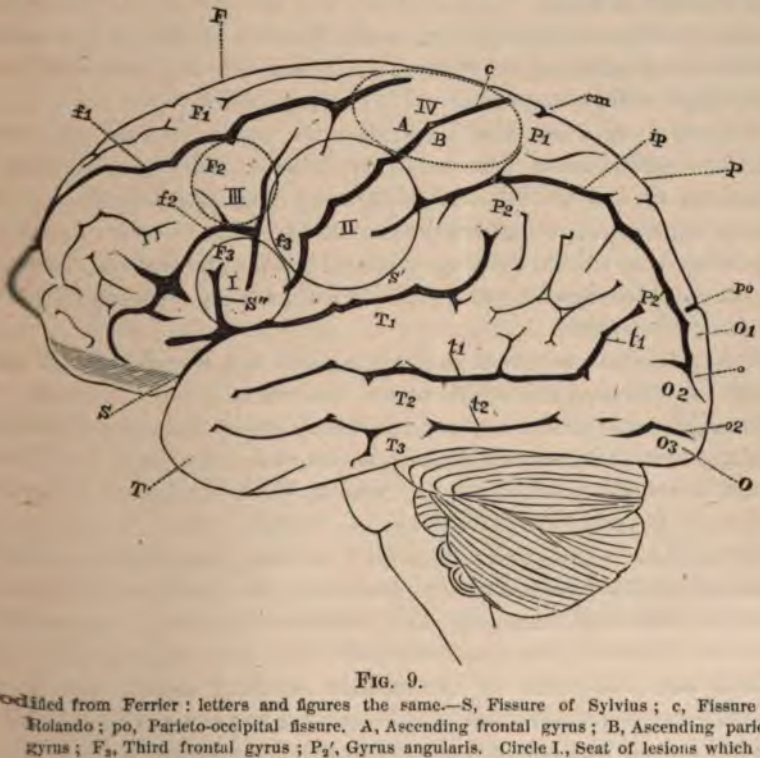

Fig. 9.

Modified from Ferrier: letters and figures the same.—S, Fissure of Sylvius; c, Fissure of Rolando; po, Parieto-occipital fissure. A, Ascending frontal gyrus; B, Ascending parietal gyrus; F_3, Third frontal gyrus; P_2', Gyrus angularis. Circle I., Seat of lesions which (on the left side) cause aphasia. Circle II., Seat of lesions which convulse or paralyze the upper extremity of the opposite side. Dotted Circle III., Seat of lesions which probably convulse or paralyze the face on the opposite side. Dotted oval IV., Seat of lesions which probably convulse or paralyze the lower extremity of the opposite side. These districts receive their blood supply chiefly from the middle cerebral artery.

post-mortem facts would seem to connect it with movements of the eyelids and upper face. Besides, the paracentral lobule is of importance to us, because we know that its destruction is followed by descending degeneration.

The cerebral convolutions are supplied with blood by branches of the anterior, middle, and posterior cerebral arteries, and an exact knowledge of the distribution of these trunks and their ramification is of the utmost importance in the study of localization of lesions, inasmuch as one of the most common pathological processes by which destruction of convolutions is caused, is embolism, or plugging of one of these arteries.

In general terms, the anterior cerebral artery supplies the inner face of the hemisphere as far back as the occipito-parietal fissure; the first, second, and (very partially) the ascending frontal convolutions.

The middle cerebral artery, or the Sylvian artery, is the most important physiologically, as it supplies all the convolutions mentioned above as concerned in the production of voluntary movements, viz., the third and the ascending frontal, ascending, first and second parietal convolutions. Easily recognized branches of the middle cerebral artery furnish blood to the third frontal (first branch in the fissure of Sylvius), to the ascending frontal, to the ascending parietal; a fourth branch extends as far as the angular gyrus, and a fifth supplies the first temporal convolution.

The posterior cerebral artery supplies the remainder of the temporal lobe and the whole of the occipital.

It might not quite be out of place to state here that, according to Charcot and Duret, the basal ganglia of the brain receive their blood through small branches which leave the great arteries very near the origin of the circle of Willis. The anterior part of the nucleus caudatus is supplied by arterioles derived from the anterior communicating artery, and from the first portion of the anterior cerebral artery. The nucleus lenticularis and the anterior part of the thalamus opticus are vascularized by branches of the trunk of the middle cerebral artery before it enters the Sylvian fissure. The larger part of the optic thalamus is supplied by vessels coming from the second portion of the posterior cerebral artery beyond the circle of Willis. Finally the inner aspects of the thalamus and the walls of the third ventricle receive branches from the posterior communicating artery, and from the first portion of the posterior cerebral artery within the circle of Willis.

To return to the arteries of the cortex. Ramifying in the pia covering the convolutions, they penetrate the nervous tissue in a peculiar manner, in the shape of long and straight branches, which supply the various layers of the cortex by means of horizontal branches, and ultimately, in small numbers, and greatly reduced in size, reach the white substances. A most important peculiarity in the superficial and deep cortical circulations is the absence of anastomosis between arteries of any great size. As to the exact amount of anastomosis there is a difference of opinion between the two original observers in this matter—both equally competent—Duret, of Paris, and Heubner, of Leipzig, The former maintains, and pathology supports him, that there is next to no anastomotic circulation upon or in the cortex (except through capillaries) while Heubner thinks that considerable branches of the great cerebral vessels open into one another upon the surface of the brain. The importance of this point for the prognosis of embolism of the cerebral arteries is enormous, and for my part I would say that I am disposed to consider Duret's statement as more applicable to practice.

I now pass to a very short account of the physiology of the cortex. My statements upon this matter will be all the briefer because an excellent and full account of the physiology of the cerebrum is accessible to all of you in Prof. Dalton's "Treatise on Physiology." Dr. Dalton himself has taken an honorable part in the researches which have, in the last five years, revealed unsuspected properties in the cortex of the brain.

First. We now know, since the experimental researches of Fritsch and Hitzig (1870), and of Ferrier (1873), that the cortex of the brain is excitable; *i. e.*, that galvanization or faradization of the cortex produces muscular movements in the body and limbs. This fact, standing out in direct contradiction to the teaching of all physiologists from Magendie and Müller, is a monumental acquisition to biological science.

Second. Very numerous researches by Hitzig, Ferrier, and a host of others, appear to have established beyond question that a certain relation exists between well-defined portions of the cortex of one hemisphere and limited muscular groups (almost individual muscles) in the opposite half of the body. The areas of convolutions whose irritation by electricity is followed by definite movements of peripheral parts have been denominated motor centres, or psycho-motor centres; and a large number of these centres have been determined by Prof. Ferrier and by Prof. Hitzig, upon the brains of dogs, cats, and living monkeys, whose brains bear a certain resemblance to the cerebrum of man. While knowing of a few opposing experiments which would seem to show that there is no constant relation between the spot irritated and the resultant movement, I am bound, by the weight of evidence and by the wonderful accord between the researches of various experimenters, to accept the facts as stated above.

Third. The excitable district of the brain is its median group of convolutions, including (as sketched upon a human brain, after experiments upon monkeys) the second, third, and ascending frontal convolutions, the ascending and first parietal convolutions. This excitable district or zone includes, as you perceive, gyri which receive their blood-supply by branches of the middle cerebral artery, with the exception of the second frontal gyrus. As my chief object is not physiological teaching, I prefer simply to enumerate the cortical centres as laid down by Ferrier, not encumbering a diagram with a representation of them.

On the posterior extremity of the third frontal gyrus, near the fissure of Sylvius, is a centre for the movement of the lips and tongue (a speech-centre according to the teachings of pathology); this is numbered 9 and 10 on Ferrier's plate. Next in order, upon the lower part of the ascending frontal convolution are centres for movements of the elevators and depressors of the angle of the mouth; numbered 8 and 7. Still higher on this gyrus is a centre for movements of the forearm and hand; numbered 6. Upon the upper two-thirds of the ascending parietal convolution are several centres for complex movements of the hand and wrist; designated by Ferrier, *a*, *b*, *c*, *d*. Much farther forward, upon the hemisphere near the great longitudinal fissure, is an extensive region embracing the posterior parts of the first and second frontal gyri, governing lateral movements of the head, elevation of the eyelids, and dilatation of the pupil; numbered 12. Immediately behind this, near the longitudinal fissure, upon the posterior extremity of the first frontal convolution, is a centre for extension and forward movements of the hand and arm; numbered 5. The posterior (inner) ends of the ascending frontal and ascending parietal convolutions contain centres (not clearly differentiated) for complex movements of the arms and legs together; numbered 2, 3, and 4 on Ferrier's plate. Finally, behind these on the superior parietal lobule is a centre for movements of the leg and foot; numbered 1.

The sensory centres of Ferrier occupy various parts of the inferior parietal lobule, the gyrus angularis, the second occipital, and first temporal convolutions. Although the experiments of various observers make it exceedingly probable that these inferior and posterior portions of the hemisphere are connected with general and special sensory functions, yet, as human pathology has so far thrown no light upon these questions, I shall hereafter confine myself to the study of the motor districts of the convolutions.

Not to weary you by the citation of the now very numerous cases in which localized cortical lesions, as tumors, abscesses, clots, softening, pressure-effects from bone or thickened meninges, have given rise to definite symptoms, in close or even almost exact agreement with the data obtained by faradizing the cortex, I shall state in a general manner the tendency of these recent clinical and post-mortem studies.

In the first place, it appears almost absolutely certain that in man a lesion involving the posterior part of the third frontal convolution (on the left side usually) causes aphasia; *i.e.*, impairment or loss of articulate speech, or even of language in general. It would seem, besides, that (1) lesions of the same part on either side of the brain produce paresis of many muscles concerned in lingual and pharyngeal movements; (2) that lesions of the anterior folds of the island of Reil, convolutions which are continuous with the third frontal, may also produce aphasia; and that (3) loss of speech may result from injury to the white substance lying between the third frontal gyrus and the basis cerebri. I believe, you observe, in a not too limited localization of the motor functions exerted in language, and would graphically represent this by the circle marked I. in Fig. 9.

In the second place, lesions limited to the inferior portions of the ascending frontal and parietal gyri have produced spasmodic and paralytic phenomena limited to the upper extremity of the opposite side. I am disposed to admit as highly probable that these parts are connected, in the healthy living man, with the various voluntary movements of the arm and hand. This zone is represented in Fig. 9 by circle II.

I am not prepared to go further in admitting pathologically proved cortical centres, but would add that there are some reasons for believing that future autopsies will locate one centre for the external facial muscles just forward of the two centres named above, viz., the region included in the dotted circle III.; and another for movements of the legs upon the upper parts of ascending frontal and parietal, as roughly indicated by the dotted oval, IV.

As regards sensory cortical centres, I have already said that we have as yet no pathological data for their study.

Having thus expressed myself about the question of cortical centres in man, I pass to the more clinical study of symptoms observed when the cortex is injured.

First. What are the symptoms of localized lesions involving the cortex alone, or the cortex and a minimum of subjacent white matter? The symptoms differ vastly in accordance with a rule laid down years ago by Brown-Séquard, according as the lesion is an irritative or destructive one; and besides, they vary according as the lesions are within the excitable cortical region (defined *supra*) or outside of it. We can clear the ground pretty safely at once by admitting that lesions irritating or destroying convolutions not embraced in the motor zone produce no symptoms at all. Large parts of the frontal, temporal, or occipital convolutions may be injured or utterly destroyed without the patient showing during life any special symptoms of organic cerebral disease. This statement is based upon the study of recent cases only, though I doubt not that in the literature of the century, numerous apparently contradictory cases might be collected. In considering this negative proposition, one proviso must be borne in mind, viz., that if the lesions of these unexcitable districts involve the dura mater, convulsions and localized cephalalgia may occur. Of this I have seen one marked example.

We are now prepared to study the symptoms of lesions in the excitable or motor zone of the hemispheres, as indicated by the various circles in Fig. 9.

1. The symptoms of an irritative lesion of these parts consist in convulsions, with or without subsequent transient paralysis; *e. g.*, such a lesion in circle III. (Hitzig's case) would give rise to spasmodic movements in the superficial muscles of the face, on the opposite side, with slight paralysis. Irritative lesions of the regions inclosed in circles II. and IV. will cause convulsions limited to, or first appearing, in the hand and arm, or foot and

leg, of the opposite side. As regards circle I., Broca's speech centre, we know little of the effects of its pathological irritation. In one case which I have placed on record, a thickening of the meninges involving the third frontal convolution of the left side produced intermittent and incomplete aphasia.

It was by the close study of the clinical and pathological aspects of cases of localized epilepsy (fingers and hands), that Dr. J. Hughlings Jackson was enabled to form his theory of motorial discharges from irritation of the cortex cerebri, and thus pave the way for Ferrier's admirable researches. Dr. Jackson must, I think, be considered, after Prof. Broca, as the founder of our present growing doctrine of cortical localizations.

2. Destructive lesions of portions of the excitable district produce paralysis in peripheral parts across the median line. The symptoms will, to a certain extent, correspond with the *precise* location of the lesions, very much as in irritative lesions; *e. g.*, embolism of the first branch of the middle cerebral artery on the left side will cause softening of the posterior part of the third frontal gyrus, with the symptom aphasia. A destructive lesion of the principal part of the motor zone on the right side will produce left hemiplegia without aphasia; but if this lesion occupy the left hemisphere, loss of speech will co-exist with the paralysis.

It must be added that secondary descending degeneration ensues after destructive lesions of the motor regions of the cortex, and that we have late contracture or rigidity of the paralyzed limbs as part of the symptom-group.

Negative characters of these cortical lesions are, preservation of sensibility in the paralyzed parts, and (except with epileptic attacks) preservation of consciousness, and incompleteness of paralysis.

In the next place, let us inquire what are the symptoms produced by diffused lesions of the cortex. As exemplified in acute meningitis, the chief symptons are delirium, convulsions, and pain; evidences of intense irritation. The coma and paralysis which follow may in some degree be caused by impaired nutrition of the cortex, but more probably by circulatory and tension-changes in the whole encephalic mass.

There is a much better disease for studying the effects of lesions of the surface of the brain, both irritative and destructive —I mean general paralysis of the insane, or, anatomically speaking, diffused, chronic, meningo-encephalitis. The affection is very common, and has been thoroughly studied, clinically and pathologically. From these studies we learn that in the first stage of the affection there occur fibrillary contractions in many muscles of the tongue, face and limbs; that speech is made tremulous and jerky; that there is over-ideation and even acute delirium; that gradually memory and judgment become impaired, and a semi-paralytic and semi-ataxic condition develops in the limbs. Later the mental faculties are abolished; a stage of dementia with occasional gleams of delirium (exalted notions), and integrity of the organic functions characterizes the disease. The attempt is now being made to show that when the meningo-cortical changes are limited to the frontal lobes the symptoms are mainly psychical; when the lesion involves the motor districts alone we observe abundant fibrillary tremors and pseudo-paralysis; and, finally, if the occipital lobes are affected sensory symptoms (hallucinations) predominate. As yet not much support has been obtained for such a distinction, which appears very tempting upon physiological grounds. It should not be forgotten, in using cases of general paralysis for the study of the question of localization, that the disease is one in which lesions exist in many parts, or almost all the parts of the cerebro-spinal axis.

The question of the localization of functions in the cerebral convolutions, and that of the possibility of diagnosticating their lesions is, as yet, in its infancy; we need numbers of exact observations to decide it one way or the other. Just now, I believe that the presumption is in favor of a positive answer; there are many facts supporting this affirmative. The clinical and post-mortem facts have just been referred to, and I shall close the lecture by recapitulating the various anatomical, physiological, and pathological evidence in favor of the existence of motor centres in the cortex.

1. Coarse anatomy enables us to trace bundles of fibres upward from the motor tract of the medulla and pons, into the internal capsule as far as the convolutions which are grouped about the fissure of Rolando. By its aid we can also trace sensory nerve fasciculi from the posterior regions of the pons to the occipital and temporal lobes, and their cortex. Such gross dissections are, however, condemned as unreliable by some authorities.

Microscopic anatomy shows that the so-called motor gyri are rich in large cells; nay, that they alone contain the "giant cells" of Betz, that is, ganglion cells, which in size and number of processes bear a remarkable resemblance to the unquestionably motor ganglion cells of the anterior horns of the spinal cord and the medulla oblongata. In the motor convolutions these giant cells are found in little clusters of three, five, or more, in a section, imbedded among the large ganglion cells of the third layer.

2. Experimental physiology teaches us that electrical irritation of this zone, and of this zone only, produces muscular contraction in parts on the other side of the median line; and, further, that this zone may be divided into a number of "centres" for various small parts, tongue, face, arm, leg, etc.* By experimentation we also learn that slicing or burning off these cortical centres produces partial paralysis of peripheral parts on the opposite side of the body, with precisely the same correspondence between centres and muscular groups as the irritative experiments demonstrate.

By the latter mode of experimenting applied to the occipital and temporal convolutions (Ferrier, H. Munk, and others), it is made highly probable that there is a certain relation between parts of these gyri and the organs of special and general sensibility across the median line.

3. Pathological anatomy (recent cases) demonstrates (*a*) that destructive lesions of the motor regions of the cortex (and of the paracentral lobule) produce descending degeneration throughout the direct cerebral motor tract extending into the lateral columns of the spinal cord; and (*b*) that there is a remarkable correspondence between certain localized spasmodic and paralytic symptoms observed during life, and lesions irritating or destroying certain definite spots in the motor zone of the cortex.

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* Still more recent researches by MM. Franck and Pitrès show that after removal of the cortex in the excitable zone, faradization of those portions of white substances which are then exposed (anterior half of the internal capsule) gives rise to similar (in kind and in distribution) movements in peripheral parts across the median line.

LECTURE VIII.

SUMMARY :—SURGICAL ASPECTS OF THE QUESTION OF CEREBRAL LOCALIZATIONS—CRANIO-CEREBRAL TOPOGRAPHY, AND ITS UTILIZATION IN DIAGNOSIS AND FOR OPERATIVE PROCEDURES.

GENTLEMEN :—The question of the utilization of the doctrine of localization in surgery remains for study. This, the most novel part of the subject, is, I think, of great present interest and of much promise in the future. Already several brilliant surgical operations have been performed upon indications derived from the newly acquired knowledge of cranio-cerebral topography. By this term we mean the determination of the relations between the external surface of the skull and the principal gyri and sulci of the brain. So little was done toward ascertaining these relations that up to 1861 the position of the fissure of Rolando relative to the coronal suture was wholly unknown. In that year Prof. Broca invented a scientific procedure for the study of the subject; he inserted pegs into the cerebral substance through holes drilled into the skull at given points, and then, removing the skull-cap carefully, was enabled to determine exactly what convolutions corresponded to the pierced regions of the skull. He thus discovered that the parieto-occipital fissure lies under the lambdoid suture, and that the fissure of Rolando slopes backward, so that its posterior extremity is placed at more than forty millimetres behind the coronal suture. Since that year the subject has been thoroughly studied by Broca, Bischoff, Heftler (1873), Prof. Turner, of Edinburgh (1874), Ch. Féré and Broca (1875), and others. Perhaps the best and most applicable of these contributions is that of Féré, and I shall follow it closely in the following remarks. It should be borne in mind that for purely anthropological purposes, the determination of the relation of gyri and sulci to certain sutures or processes of the bare skull is sufficient; but that for use in the regional diagnosis of cerebral injuries, and in practical surgery, the cranial landmarks should be such as are easily determined upon the scalp and face of the living man. The *résumé* of cranio-cerebral topography which I offer for your guidance is based upon the latter principle of study.

As shown in Fig. 10, I shall sketch the situation of the principal convolutions, fissures, and central gray bodies of the cerebrum upon an outline figure of the profile of a skull. The skull is represented as resting upon a peculiar plane, one passing under the condyles of the occipital bones and the alveolar processes of the superior maxillæ—the alveolo-condyloid plane of Broca. Upon this horizontal line, which can be determined with reasonable accuracy in the living human being, we erect other lines and measure distances which enable us to solve almost the whole problem.

1. A vertical line (A) drawn from the alveolo-condyloid plane through the external auditory meatus upward will pass through, or very near to, the bregma or line of junction of the frontal and parietal bones at the vertex; it passes through the anterior (lower) extremity of the fissure of Rolando.

2. If from the upper end of this vertical line A, we measure a distance of 45 mm. backward toward the occiput and draw a descending vertical line (1–2), we mark out the location of two most important parts of the cerebrum, viz., the posterior extremity of the fissure of Rolando, and the posterior limit of the thalamus opticus in the hemisphere [at *c*)].

3. To conclude with the occipital end of the skull; if we can make out with the finger the lambdoid suture at the median line, we thus learn the situation of the subjacent occipito-parietal suture, which separates the parietal and occipital lobes.

4. The last vertical line worth noting is one drawn at a distance of 30 mm. forward of the auriculo-bregmatic line. This vertical line (3–4) will pass through the middle fold of the third frontal convolution (just forward of the speech centre), and will also indicate the anterior limit of the central cerebral ganglia, viz., the head of the nucleus caudatus in the hemisphere [at (*d*].

5. The upper level of the central cerebral ganglia may be quite exactly indicated by a horizontal line drawn at a distance of 45 mm. below the surface of the scalp at the bregma (or 35 below the surface of the bare skull at the same point). This line (7–8) also extends across the middle regions of the motor district of the convolutions, containing centres for the face and upper extremities.

6. The external angular process of the frontal bone, not difficult to define in the living subject, is the starting-point of another horizontal line (5–6), whose posterior extremity passes a little below the lambdoid suture. Upon this horizontal line we can, by measurement, determine the location of certain parts. Thus, at a distance of 18 or 20 mm. behind the external angular

process lies the folded part of the third frontal convolution (*a*). This point in many heads will correspond with the vertical line 3–4.

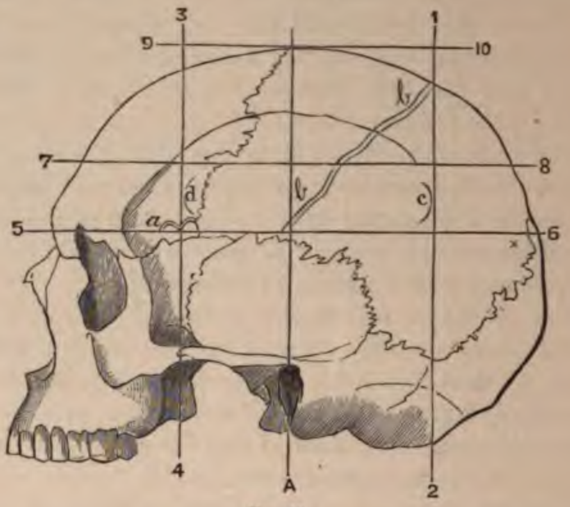

FIG. 10.

Outline of skull resting upon the alveolo-condyloid plane of Broca; modified from Topinard (*Anthropology*). Vertical line A, or auriculo-bregmatic. Line 9-10 drawn parallel to the plane of Broca. Upon this line, at a distance of 45 mm. posterior to the bregma, a vertical line, 1-2, will pass through the upper (inner) end of the fissure of Rolando, *b b*, and through the posterior extremity of the thalamus opticus, (*c*). A third vertical line, 3–4, drawn at 30 mm. forward of the bregma, will pass through the fold of the third frontal gyrus, *a*, and through the head of the nucleus caudatus (*d*). The horizontal line 7–8, at 45 mm. below the bregma (scalp), indicates the upper limit of the central ganglia. The third horizontal line 5–6, passing through the external angular process of the frontal bone and the occipito-parietal junction, approximately indicates the course of the fissure of Sylvius, and serves for measurements. At 18 or 20 mm. behind the ext. ang. process on this line is the speech centre of Broca; 5 to 8 mm. behind the intersection of 3–4 and 5–6, is the beginning of the fissure of Sylvius, and at 28 or 30 mm. behind this intersection is the lower end of the fissure of Rolando, *b b*, placed a little too far back in the cut. At × (near 6), near the median line, is the location of the occipito-parietal fissure.

7. The situation of the fissure of Sylvius may be approximately ascertained in the following manner: Its middle portion extends horizontally, almost under the upper part of the squamous suture, which in the living subject is to be found a little below the horizontal line 5–6. The anterior extremity or beginning of the fissure of Sylvius is a little below this horizontal line, at a distance of some 5 to 8 mm. posterior to the intersection of 3–4 and 5–6, and consequently about 22 or 25 mm. anterior to the auriculo-bregmatic line A. Lastly, according to Turner, the parietal eminence almost always overlies the supramarginal gyrus (P_2, Fig. 9), consequently the posterior extremity of the fissure of Sylvius is likewise in this vicinity.

8. The angular gyrus is to be found below and behind the parietal eminence, a little above the horizontal line drawn from the external angular process (5–6).

9. The anterior (lower) end of the fissure of Rolando lies at a distance of 28 or 30 mm. behind the line 3–4, and a little above 5–6. It is therefore a few mm. anterior to the vertical line A.

The application of these data to practical medicine and surgery is quite obvious.

In medical cases, when tumors of the skull develop externally, we may determine by cranio-cerebral topography whether their extension inward can give rise to the motor symptoms which are present (convulsions and paralysis), according as the excitable districts of the brain are threatened by extension of the growth from the inner surface of the skull, or not.

In surgery, the utility of cranio-cerebral topography is much greater.

For example, as far back as 1871, Professor Broca was able to correctly diagnosticate an abscess of the left third frontal convolution, and was successful in trephining directly over it. In 1876, Proust and Lucas-Championnière successfully trephined the skull, in two cases, for the removal of fragments which were compressing the ascending frontal and parietal convolutions, and causing paralysis.

In the case of a patient who is paralyzed on one side of the body, after an injury to the skull, the following considerations might justify, or contra-indicate an operation for the removal of bone, of blood, or of pus:

1. If the hemiplegia (or hemispasm) be very complete, it is probable that the injury to the brain is considerable in extent, and extends deeper than the special centres for the face, arm, and leg.

2. If the externally evident injury be over non-excitable convolutions, and the paralytic or spasmodic phenomena be marked, it is more than probable that the brain is torn or compressed at other points than under the seat of injury, and an operation is contra-indicated.

3. If the paralytic or convulsive symptoms be on the same side as the evident cranial injury, it is probable that there are cerebral lesions on the other side produced by *contre-coup*, hence interference will be undesirable.

4. Even if a cranial injury be directly over excitable convolu-

tions, if the resulting paralysis or convulsions be accompanied by marked anæsthesia, an operation cannot be expected to do much good, because the presence of anæsthesia makes it highly probable that the white substance of the hemisphere (posterior half or third of the internal capsule) is involved in the lesion.

5. A favorable indication for trephining is when aphasia supervenes immediately, or in a few weeks after an injury to the skull in the region of the left third frontal convolution (see Fig. 10). It is extremely likely, in the first case, that a clot or spicule of bone will be found compressing or lacerating the centre for speech ; in the second, that an abscess has formed in the same part (Broca's case).

6. Another combination of symptoms which makes an operation desirable, and holds out a hope of its being successful, is when an injury to the skull over the fissure of Rolando on one side is accompanied by slight hemiplegia, or by paralysis of the face, or arm, or leg, or any two of these parts combined, on the opposite side of the median line, without anæsthesia. Under such circumstances, the probabilities almost amount to certainty that the centres for (see Fig. 9) the face, arm, and leg are separately or collectively involved in the lesion (cases of Proust and Lucas-Championnière).

7. A contra-indication to operative interference, even in apparently favorable cases, would be symptoms of basal lesions, such as palsy of cranial nerves, neuro-retinitis, Cheyne-Stokes respiration, and vomiting.

THE DIAGNOSIS OF PROGRESSIVE LOCOMOTOR ATAXIA.*

GENTLEMEN:—The diagnosis of sclerosis of the posterior columns of the spinal cord, or, clinically speaking, of progressive locomotor ataxia, is one which ought to be made readily by all practitioners; and yet the experience of specialists is to the effect that mistakes in this matter are very common. Consequently it may be worth our while to consider the means of correctly diagnosticating this terrible disease, by a short and clear study of its capital symptoms, and their grouping.

It has seemed to me that the causes of mistaken diagnosis are two-fold:

First—Medical men generally do not fully appreciate the value of the symptom fulgurating pains, as indicative of disease in the posterior columns of the spinal cord. This want of clear understanding leads to calling the first stage of locomotor ataxia by such names as "rheumatism," "neuralgia," etc.

Second—Practitioners have in some manner acquired an exaggerated notion of the value of another symptom, viz., staggering or falling when the patient's eyes are closed. This second error conduces to calling a variety of morbid states by the name of an incurable disease. Even the symptom ataxia, I hope to show, is not to be accepted as pathognomonic of the disease in question.

Having these views of the reasons why one of the most definite of spinal diseases is so often ignored, or wrongly attributed to the patient, you may understand why I seem to make my clinics the means of studying semeiology in detail and critically. It has seemed to me, for several years, that among the many desiderata in the medical curriculum, none is more important and urgent than that students and practitioners should devote themselves more to analytical semeiology. This exhaustive study of symptoms is to practical medicine very much as general histology is to the great sciences of anatomy and physiology. It is a matter of constant surprise how seldom symptoms (especially nervous symptoms) are determined with scientific accuracy by physicians; and yet the symptoms of disease are the data from which we reason inductively to a diagnosis. If all the symptoms be carelessly observed, how insecure must the conclusion be. And if one symptom is erroneously determined or interpreted, the whole fabric of diagnosis, in many cases, falls to the ground like a house of cards. As an incentive to further analytical study of the symptoms of disease, I may add, without exaggeration, that, with our present knowledge, the most exact and conscientious examination of a patient sometimes fails to furnish the elements for more than what I am in the habit of calling a diagnosis of probability.

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* From A Series of American Clinical Lectures, edited by E. C. Seguin, M.D. Volume III., No. XII. New York, Nov. 13, 1878.

But to return to our subject. I propose studying it in the following manner: First—Enumerate the symptoms of progressive locomotor ataxia, and classify them into those which are important for diagnosis, and those which are unessential. Second—Group these symptoms into the recognized stages of the disease, and illustrate by cases the diagnosis in each stage. Third—Consider the differential diagnosis between locomotor ataxia and a few conditions which resemble it, and which are sometimes taken for it.

I. The symptoms of progressive locomotor ataxia are the following:

(*a*) Symptoms of real utility in diagnosis:

Fulgurating pains.
Hyperæsthesia.
Anæsthesia.
Ataxia.
Paralysis of ocular muscles.
Absence of tendon reflex.*

(*b*) Other symptoms, not essential to diagnosis:

Atrophy of the optic nerves.
Pupillary changes.
Disorders of various cranial nerves.

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* Absent tendon reflexes are placed low, by the author, because the same symptom is also present in myelitis anterior, diphtheritic myelitis, and some neural diseases, besides being apparently the normal state in a few healthy people.—[R. W. A.]

Numbness in lower and upper extremities.
Peculiar sensations under feet.
Staggering or falling when eyes are closed.
Sense of constriction about limbs or trunk.
Paresis of bladder and rectum.
Increase and decrease of sexual excitability.
Rectal, vesical, gastric and laryngeal "crises."
Arthropathies.
Dementia.
Muscular atrophy.

The diagnostic symptoms are worthy of a full description, since their clear appreciation is so important:

1. By fulgurating pains we mean abnormal sensations having the following characters: They are painful sensations, varying in degree from the feeling produced by the prick of a needle to the most excruciating agony. Patients describe quite a variety of these fulgurating pains; some are like needle-pricks, others like knife-cuts, perpendicularly or longitudinally inflicted; others resemble the crushing of muscles and bones, yet others are as if a given piece of nerve were pulled or rubbed, and some are like the working up and down of a bolt or a wire in a given space in the limb. However varied in character, these pains are always *sudden*, *localized*, *repeated* and *vagrant*. They are so *sudden* in their appearance that a strong man is surprised into a loud exclamation of pain by their appearance, while feeling otherwise well; hence the term fulgurating pain. They are *localized* in several senses. First, they make their appearance always (?) in the lower extremities, at any rate in those parts which are later to become ataxic. Second, they are localized in circumscribed regions of tissue, usually the skin. In the vast majority of cases rounded or oval spots in the foot, thigh or leg, are the seat of fulgurating pains. In other instances a circumscribed mass of deeper tissue (muscle or bone, or joint apparently) is the seat of tearing or crushing pain. These pains are *repeated*; that is to say one patch of skin, or mass of deeper tissue, is the seat of a succession of stabbing or crushing pains, occurring every few moments for minutes, hours or days. For example, a spot the size of a silver dollar upon the instep or calf may be the theatre of an agonizing paroxysm of pain; innumerable acute darts of pain appearing in the region for one

night or twenty-four hours. Lastly, fulgurating pains are *vagrant*. No one nerve trunk is their habitual seat, but any portion of the extremities may be visited by them. Patients usually give up in despair the attempt to show you every spot in which they have suffered; and they generally indicate as foci of pain the heel, instep, and thigh. If we consider the pathological anatomy of the disease, we easily understand why almost every sensory nerve of the affected extremities should at various times react to the irritation to which its rootlets are subjected in the posterior radicular columns.

You perceive that by the above characters you may distinguish fulgurating pains from a variety of others. For example, sciatica, or any neuralgia of the extremities, is characterized by the recurrence of pain along the nerve trunk, or along some of its branches—the patient can usually mark out with his forefinger the distribution of the affected nerve. In rheumatism the pain is usually dull, deep-seated, affecting muscles or articulations; it often involves numerous muscles or joints at one time, and is made worse by exertion. As regards the influence of weather, both fulgurating and rheumatic pains are apt to occur or increase in severity just before storms or sudden depressions of the barometer. The pain caused by vertebral caries, or by spinal tumors, is localized, and often can be referred to a spinal nerve trunk and its distribution. In poliomyelitis anterior, and in muscular atrophy there may occur severe pains, but they more resemble neuralgic pains as above described.

2. The hyperæsthesia observed in locomotor ataxia is not peculiar in kind, but in distribution. It consists in ordinary over-sensitiveness of the skin, tenderness if you please, limited to the patches of skin which are the seat of fulgurating pains. For example, if an oval spot about two inches in diameter on the anterior region of the thigh be the seat of stabbing pains for a few hours, it becomes exquisitely tender, and remains so for some time after the termination of the paroxysm. In ordinary neuralgia the hyperæsthesia is very different; we find a number of tender "points," not patches, some at the places where the nerve trunk becomes accessible to pressure, others where its branches approach a cutaneous distribution; there are nearly always two or more points in a neuralgic district.

3. Anæsthesia occurs in two ways in locomotor ataxia. At

an early period it is claimed that careful observation will discover limited, and usually laterally symmetrical patches of skin which are partially or wholly devoid of sensibility. Possibly these patches represent regions where fulgurating pains have been repeatedly experienced. Later in the disease a progressive impairment of sensibility is developed in the feet, and ascends; it may ultimately become complete throughout the lower (and upper) extremities. The chief peculiarities of this anæsthesia are, the predominance of analgesia or failure to perceive painful impressions, and retardation in the transmission of what impressions are perceived. This retardation I have often found, and have known an interval of more than one minute to occur between the moment of pricking a patient's foot, and that of his saying that he felt it. Intervals of ten or twenty seconds are frequently found. I intend saying more of this anæsthesia when I come to speak of differential diagnosis.

4. Ataxia of movement, a variety of inco-ordination, is justly held to be a most valuable symptom of progressive locomotor ataxia. Yet it is by no means pathognomonic as the disease (or lesion) may exist and the patient be incurable without a trace of ataxia being present; and conversely, ataxia may be exhibited by patients who have not systematic sclerosis of the posterior columns. An exact delineation of ataxia seems to me as important to attempt as that of fulgurating pains. By ataxia I understand a disorder in voluntary movement caused by want of harmony in the action of antagonistic muscles. The result is, that in doing a willed act, as walking, or putting the index finger on a given spot, there occur certain oscillations of the member used, which oscillations are the result of inharmonious automatic action of various muscular groups, extensors, flexors, abductors, and adductors. In the lower extremities the extensor and abductor muscles usually predominate, hence the jerky, stamping walk of tabetic patients. Several peculiarities are worth noting. Ataxic movements are not observed while at rest; they become evident when volitional acts are attempted. In chorea we have a form of inco-ordination in which various muscular groups contract involuntarily and at irregular times, but as much when the patient tries to keep still as at any time. Besides, in chorea, it cannot be said that antagonistic groups of muscles are affected, as the muscular contractions attack any and all muscles in the most capricious manner. Again, ataxic

movements are not rhythmical, which serves to distinguish them from the movements observed in paralysis agitans.

The presence of ataxia may be determined by the following methods of examination: When the patient is sitting or lying quietly no disorder is visible. On bidding him try to walk, if still able to stand, he starts off with his feet thrown outward and forward in a very exaggerated manner, and the heel is brought down forcibly. At the same time he staggers or oscillates like a drunken man, but this is not from ataxia. If the patient be unable to stand alone, upon being supported by a person on either side he will make the same punchinello-like movements of the legs in lieu of regular walking movements. In both degrees of disability the extent of irregular movements is much increased if the patient's eyes be closed. If the patient be placed upon a couch and told to extend and abduct one of his legs so as to bring his great toe against the observer's forefinger, large oscillations will occur, and the attempt may wholly fail. Again in trying to do this, or in simply trying to raise the leg, such enormous extensor and abductor action may take place as to cause the limb to strike persons standing by the bed. While recumbent, if the patient's eyes be closed and he be told to place the heel of one foot upon the patella of the opposite leg, he will do it only after great aberrations, or wholly fail.

If we suspect ataxia in the upper extremities we bid the patient place the end of his forefinger upon a small object, such as our own forefinger, or the end of his nose. If there be marked ataxia the object will only be reached after a number of misses or oscillations of the whole forearm and hand. If he try to touch his own nose he may only succeed after having poked his finger into his mouth or eyes, or upon the cheek. Upon attempting the same action with eyes closed the difficulty is greatly increased, and may actually be insuperable. All actions requiring co-ordination, as eating, writing, dressing, etc., become difficult or impossible. In all this disorder we observe no tremor or choreic jerks.

Mere muscular strength is nearly or quite preserved in arms and legs.

Paralysis of various ocular muscles requires no description. Let it suffice to say that the third nerve is the one usually affected, though the abducens and patheticus are also paralyzed in some patients. The importance of the symptom lies in its frequency, and its accompanying or even preceding the fulgurating pains of the first stage of the disease. So true is this that a good many patients first consult an oculist for some trouble about the eye, and are by him referred to neurologists.

II. Let us now pass on to the diagnosis of progressive locomotor ataxia in its three generally recognized stages:

(a) The first stage of the disease may well be designated, the *stage of fulgurating pains*. In order of frequency and importance these are the symptoms which characterize it:

Fulgurating pains.
Localized hyperæsthesia.
Diplopia from strabismus.
Ptosis from palsy of third nerve.
Small pupils.
Unequal pupils.
Numbness and slight anæsthesia of feet.
Sexual excitement.
Seminal emissions.
Paresis of the bladder.
Diminished tendon reflex (tested at the knee).
Impaired sight from atrophy of optic nerves).
Slight arthropathies.
Localized anæsthesia.
Absence of paralysis or ataxia in the limbs.
General health excellent.

Of this long list only the fulgurating pains and ocular paralyses are of great importance, the former only are absolutely indispensable to the diagnosis. In some cases, for months and even years, no symptoms are present except fulgurating pains, and I think that from them alone, if we deal with an ordinarily intelligent patient, the diagnosis ought to be made. How many men have come to me with the story that during years of pain preceding any disorder in movement, their family physician had told them, "Oh, its nothing but rheumatism."

Very small pupils with even rare fulgurating pains make up quite a clear symptom-group; and if to these we have added in a few months ptosis or internal strabismus, the case is very positively one of sclerosis of the posterior columns. Once in a great while we meet with the early combination of atrophy of the optic nerves and fulgurating pains. I have seen examples of this unusual early stage.

The following histories illustrate the first or neuralgic stage of sclerosis of the posterior column.

CASE I. Prolonged first stage. Male, aged 57 years; an artist. Comes for advice about a "neuralgia" of long standing. For twenty-seven years has had severe pains in his lower, and since two or three years also in the upper extremities. Patient describes these pains as sudden, sharp, teasing, sometimes of atrocious severity, occurring in spots or patches of round or oblong shape. These pains recur in one spot for some time, varying from a few minutes to hours and days. In the course of these many years has had foci of pain in nearly every part of the lower extremities, more especially near the knees and ankles. In the last few years the intervals between paroxysms have become shorter, and the pains have grown more severe. There is now mydriasis of the right eye, a condition which has existed thirty years, without diplopia. A mere trace of numbness has made its appearance in the legs, detected only at times by rubbing the skin. Painful spots are hyperæsthetic during and after paroxysms. In the last few years urine slowly passed. Floor or ground feels normal under feet; no difficulty in walking. Examination shows dilatation of right pupil; no diplopia, or changes in the optic nerves; no ataxia of upper or lower extremities; very slight staggering when patient attempts to stand with eyes closed. The soles of the feet show a trace of anæsthesia to anæsthesiometer. Reflex from ligamentum patellæ lost; no paresis. I am disposed to believe that this is the longest first stage of posterior spinal sclerosis on record.

CASE II. Prolonged first stage: arthropathies. Male, aged 32 years. Sent to my clinic by Dr. C. Williams. Characteristic pains in lower extremities for twelve years; spots of pain hyperæsthetic at time of attack. Slight numbness of feet; swelling of both knees in last two years; no difficulty in locomotion. Examination shows moderate anæsthesia and analgesia in feet and legs nearly to knees; absence of knee tendon-reflex; double chronic arthritis of knee-joints with crepitations. Careful testing with eyes open or closed reveals no ataxia or staggering.

In the vast majority of cases, as exemplified in the succeeding cases, the duration of the first stage is from one to four years.

(*b*) The beginning of the second stage is characterized by the appearance of ataxic movements amid a large number of other symptoms. This may be called the *ataxic stage.* The chief symptoms are, in order of importance:

Ataxic movements.
Fulgurating pains.
Localized hyperæsthesia.

Ocular paralyses.
Numbness and other dysæsthesiæ.
Anæsthesia.
Staggering with closed eyes.
Failure of sexual power.
Absence of tendon-reflex.
Rectal and vesical paresis.
Gastric crises.
Laryngeal "
Vesical "
Severe arthropathies.
Amaurosis.
Complicating common transverse myelitis.
Spinal congestion.
Paralytic dementia.
Vesical catarrh.
Preservation of mere muscular force.

The exact grouping of the above symptoms at a given period varies infinitely in a series of cases. The all-important symptom is ataxia. Yet ataxia without some of the above accompaniments, notably without fulgurating pains, does not indicate systematic sclerosis of the posterior columns of the spinal cord, though it may mean that there is *some* disease of these columns, as, *e. g.*, in disseminated nodular sclerosis, no diphtheritic ataxia, etc. As regards the distribution of the ataxia, it is paraplegic in the vast majority of cases for all time, or for a long period. The upper extremities may be affected very severely while the patient is able to stand and take a few steps with the help of a cane, whereas in other cases the patient may die after having been many years bedridden without any extension above the waist taking place.

The following are abbreviated histories of patients first seen in the second or ataxic stage of posterior spinal sclerosis. They illustrate the varied grouping of symptoms.

CASE III. First stage passing into the second. Male, aged 35 years. During the autumn of 1877, and since, has had moderately severe fulgurating pains in thighs, legs and feet. These are short, sharp, darting pains recurring in one spot for minutes or hours; the last paroxysm was upon the left instep and lasted twenty-four hours. During the spring and summer has thought that his eyes were growing weaker. Numbness has appeared in the sole of both feet only in the last two weeks. Once or twice urine has escaped involuntarily. No diplopia, or impairment of vision. Patient standing with closed eyes oscillates a little; his walk is a little stamping but not clearly ataxic. On the soles of the feet the æsthesiometer shows slight anæsthesia; pricking and touching are well felt. No reflex action from sole or ligamentum patellæ. Strength at knee-joints, and grasp normal. Is generally weak and anæmic.

In this case the first stage is about to pass into the second, or ataxic stage, after having lasted only about a year.

CASE IV. Developed second stage with ocular paresis. Male, aged forty-four years. Was well until the summer of 1876, when there occurred dizziness and a tendency to go one-sided in walking. In the course of the next few months legs became weak but not numb, and in the summer of 1877, eyes became affected. Patient denies having had pains, but says that he has "rheumatism," and proceeds to describe regular fulgurating pains in various spots in the lower extremities; shooting pains in one spot for hours or days These spots were hyperæsthetic. Has had such pains for four years. When he consulted an oculist in summer of 1877, had diplopia, which has continued. For months has staggered when closing eyes (or when washing face); and feet have also been numb. It seems to the patient as if he were walking on cotton or india-rubber. No vesical symptoms; no symptoms in hands. Examination shows slight diplopia when looking outward to the right (paresis of right abducens), upper extremities strong and well co-ordinated. The walk is highly characteristic; the feet are kept apart, body swaying, legs jerked forward and outward, and heel brought sharply down at each step. Closing eyes makes standing impossible; looking up at ceiling aggravates walk. Soles of feet are much anæsthetic; sensations blunted and retarded. Reflex from soles abnormally great; no tendon-reflex at knees.

CASE V. Fully developed second stage; ocular symptoms, gastric crises. A female, aged fifty-two years, observed in the Presbyterian Hospital, January, 1873. Admitted to the hospital on November 16th, 1872; gave the following history: eighteen months previously had a first attack of severe vomiting, recurring every two or three hours, and extending over a period to thirty-six hours. Similar seizures recurred almost punctually every three months until last March. About ten months ago, after the third attack, she experienced sharp, shooting pains in her lower extremities from the hips down, continuing more or less actively for about three weeks. Although patient was weak, yet she could walk well enough at that time, but not long afterward legs became feeble. Last spring, and since, the vomiting recurred more frequently, less violently, and irregularly. Two months ago (September, 1872), she noticed that the lid of the left eye could not be fully raised. Just previous to this she had experienced shooting pains in the upper extremities; pains just like those which had occurred in the lower extremities. The pains in the arms lasted about two weeks. From time of admission to January 1st, 1873, patient was treated by Dr. Wynkoop for nausea and frequent vomiting. On taking the service I examined Miss L., and determined the presence of the following symptoms. There is ptosis on the left side, the pupil is a little dilated, and there is some external strabismus. Does not converge well with right eye. Slight anæsthesia of the second and third branches of the trigeminus, (side not stated). Grasp of hand good; with eyes closed patient has difficulty in placing forefinger on end of nose, *i.e.*, there is ataxia of the upper extremities; more on the left side. Slight tactile anæsthesia of fingers and hands. Lower extremities are not, strictly speaking, paretic. In recumbent posture slight inco-ordination of legs. Later the vomiting was controlled; hysterical symptoms of various kinds set in (so marked as to lead some of the medical staff to doubt the correctness of my diagnosis*), and, finally, on April 14th, the patient died suddenly of cerebellar hæmorrhage. A post-mortem examination, supplemented by microscopic study of specimens, showed that the posterior columns of the spinal cord were extensively sclerosed.

(c) The third stage may be said to begin when the anæsthesia and ataxia are so great as to render the patient perfectly unable to stand, or to "use his legs," as he terms it. This might aptly be called the *pseudo-paralytic stage.*

In this terminal period we may have any of the following symptoms in various groupings, or even all of them:

Fulgurating pains.
Ataxic movements.
Absolute anæsthesia.
Loss of sexual power.
Rectal and vesical paresis.
Paralysis of ocular muscles.
Amaurosis.
Deafness.
Various "crises."
Severe arthropathies.
Disorganization of large joints without pain.
Seeming paralysis of the extremities from anæsthesia, (and loss of muscular sense?).
Dementia.

Mere muscular power probably preserved. Electro-muscular contractibility is preserved. Reflex movements reduced or abolished.

As complications:

Cystitis and pyelo-nephritis.
Pulmonary phthisis.

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* See page 180; also *Archives of Electrology and Neurology*, N. Y., May, 1875.

Muscular atrophy.
Transverse myelitis, etc.

What is remarkable and characteristic in such cases is that the helpless patient whose legs fly about in the wildest manner when he attempts a voluntary movement, or, who, because of absolute anæsthesia knows not how to guide his movements, can, for a few moments, show great strength of resistance to flexion at the knee-joint, or a nearly normal grasping power in his hands. In some cases, however, the patient is so completely isolated from his extremities by anæsthesia, so ignorant of their existence and whereabouts, that he can not move them. Yet, post-mortem evidence shows that the direct motor tract from the brain to the muscles is intact, and reasoning leads to the conclusion that there is at no time a true paralysis in the uncomplicated disease.

III. Differential diagnosis. This needs be made from a number of conditions. When defining the fulgurating pains, characteristic of the first stage, I pointed out to you how they may be distinguished from purely neuralgic and rheumatic pains. The diseases which are often miscalled locomotor ataxia are the following:

1. Hysterical paraplegia of incomplete degree. The legs in this condition exhibit extreme anæsthesia, and if the patient be told to stand and then close her eyes she will oscillate and fall if not supported. Tested in the recumbent posture or by walking no ataxia can be discovered, and the history of the case does not reveal the previous occurrence of fulgurating pains.

2. Diphtheritic ataxia. In 1864, Prof. Jaccoud, of Paris, called attention to the occurrence of ataxia movements in certain patients who were said to have diphtheritic paralysis. I saw a remarkable case of this sort last winter in an adult who was supposed to have locomotor ataxia. He walked precisely like a tabetic patient, jerking his legs outward and forward, bringing his heel down. But in such a case we can make out an acute development; and if the eyes are affected it is in a very different way from that seen in locomotor ataxia; in diphtheritic cases the ciliary muscle alone is paralyzed, causing loss of accommodation and wide pupils. Negatively we fail to get an account of fulgurating pains, anæsthesia of the feet, peculiar vesical symptoms. My patient recovered in a few weeks.

3. Paralytic dementia. In this disease the walk sometimes becomes almost ataxic, or at any rate a good deal of stamping with the heel is observed. In a few cases also, minor fulgurating pains occur. The distinction is founded upon the different grouping of symptoms and the unimportant nature of the pains. Dementia (gradual diminution of mental power), with more or less marked exalted notions, irregularity of the pupils, and tremulous, jerky speech are the prominent symptoms. Still it it must be borne in mind that there is a *bona fide* relationship between the two affections, shown in an ascending and a descending manner. In the ascending form true locomotor ataxia terminates with symptoms of paralytic dementia—of this I have seen at least four cases. The descending form has been alluded to at the beginning of this section. In the former case the spinal symptoms are primary and most important; in the latter the symptoms of dementia command our attention and govern the diagnosis.

4. Disseminated nodular spinal sclerosis. In this rare disease we do observe ataxia; but it is of a grosser and less symmetrical sort. One extremity alone may be affected. Fulgurating pains are absent, or if they have occurred, it is only in a special region; there is true paralysis in many parts, and the paralytic ocular symptoms are wanting. Besides, if the disease (as is usual) invade the medulla and brain, there are superadded exceedingly characteristic symptoms, viz.: interrupted or syllabic speech, nystagmus, hallucinations and delusions.

In the cerebral form of disseminated nodular sclerosis we have ataxic movements, or ataxic tremor, without marked paralysis, but with cerebral symptoms, and without fulgurating pains, anæsthesia of the soles of the feet, sexual and vesical symptoms.

5. Chronic, transverse or diffused myelitis. It must seem strange that this disease should be confounded with locomotor ataxia; but such is the fact. I have had quite a number of supposed cases of sclerosis of the posterior columns brought to me here and to my office, and the physicians in charge of the cases seemed much astonished at my diagnosis. The mistake turns wholly upon the exaggerated notion which practitioners entertain of the value of the symptom staggering with closed eyes. Patients with myelitis have numb and anæsthetic feet and legs, and when they are made to stand with eyes closed

they oscillate and fall, just as do patients with hysterical paraplegia.

I would now repeat for the hundredth time, that staggering or falling with closed eyes is present in many affections of the nervous system, central and peripheral, and that it is not characteristic of any one disease. It simply indicates, in most cases, that the feet are anæsthetic; in a few instances it cannot be explained in this way, and may be due to the loss or impairment of that questionable form of sensibility, the muscular sense.

Several years ago, in the early days of this clinic, I brought into the amphitheatre a healthy young man, whose soles had been frozen with ice and salt. He walked fairly well with his eyes open; but when they were closed, he oscillated a great deal, and was in danger of falling.

Finally, gentlemen, I would remark that the recorded cases of cure of locomotor ataxia will not stand the test of the methods of diagnosis detailed above; and their publication has not led me to abandon the opinion, held by all authorities I believe, that sclerosis of the posterior columns is an incurable disease at the present time.

REPORT ON ACONITIA IN THE TREATMENT OF TRIGEMINAL NEURALGIA.*

The annual meeting of the Therapeutical Society was held October 11th, 1878, Dr. J. R. Leaming, President, in the chair.

The Committee on Neurotics, through its Chairman, Dr. E. C. Seguin, presented the following report:

GENTLEMEN: I have the honor to submit the following brief preliminary report on the efficacy of the aconitia of Duquesnel in trigeminal neuralgia.

This matter was made a subject of study by the Committee on Neurotics early in this year, chiefly because of the wide circulation of Prof. Gubler's statement that aconitia was almost infallible in trigeminal neuralgia. His original article appeared in the *Gazette Hebdomadaire* for February 9th, 1877; and good abstracts were published in the *American Journal of the Medical Sciences* for April, and in the *Practitioner* (of London) for August, 1877. In his *Leçons de Thérapeutique,* Paris, 1877, Prof. Gubler has already stated his belief that aconitia was destined to be a very valuable remedy.

It may be interesting to recall the fact that, writing in 1874, Dr. H. C. Wood, of Philadelphia, in his *Treatise on Therapeutics,* had said that aconitia should never be exhibited internally. Drs. August and Theodore Husemann, in their admirable work, entitled *Die Pflanzenstoffe* (Berlin, 1871), gave a full account of the preparation, and the chemical and physiological properties of aconitia. They do not mention Duquesnel's preparation, though it was made in 1864.

Aconitia was extracted from aconitum napellus by Geiger and Hesse in 1833. This aconitia was amorphous, and probably impure, as are also the preparations now furnished by druggists under the names of Merck's, Hottot's and Morson's aconitia. Of these the last is considered the purest and best.

Duquesnel's aconitia in crystals, although discovered in 1864, has been in use, apparently, only for the last seven years—since the experimental researches of Gréhaul and Duquesnel in

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* Reprinted from the New York *Medical Journal,* December, 1878.

1871. The only sample of Duquesnel's preparation in this city to my knowledge is that held by Dr. Neergaard, the distinguished pharmacist. With the chemistry and pharmacy of aconitia we have little or nothing to do, but an epitome of its physiological effects may not be out of place.

From Husemann's, Wood's and Gubler's accounts the following may be stated with reference to the effects of this powerful alkaloid upon the animal organism.

It paralyzes the sensory nervous system at its peripheral extremities, and (probably) at its perceptive centres.

It paralyzes the heart directly, and by way of the vagus nerve. The pulse-rate is reduced. It lowers the arterial tension. It is doubtful if it affects the motor-nervous system directly. The subjective sensations of a patient who is fully under the influence of aconite or aconitia are: Numbness and tingling of the skin and mucous membranes, especially in the hands and tongue, a sense of chilliness and faintness, and indefinable nervousness.

The doses of aconitia vary very much, according to the preparation used and according to the idiosyncrasies of patients. In general terms the initial dose of all three kinds—Morson's, Hottot's and Duquesnel's—may be 0.0005 gramme given twice or thrice a day. Prof. Gubler states that the dose of amorphous aconitia may be gradually raised to 0.005 gramme. He states that Duquesnel's crystallized aconitia is much stronger, and that we must be more careful in dosing it.

In my own practice I have used great caution in prescribing Duquesnel's aconitia. My formula (first used last winter) is as follows:

℞.	Aconitiæ (Duquesnel's),	.006
	Glycerineæ,	
	Spts. vini rect.,	āā 4.
	Aq. menthæ pip.,	ad 62.
	M. One teaspoon=about .00045.	

S. A teaspoonful two or three times a day on an empty stomach.

In some cases I have used .008 or even .01 aconitia in the same formula. In this combination the solvent is the alcohol. The effects of various doses of aconitia upon our patients will be stated in the remarks which follow the relation of the following cases observed by your committee:

CASE I. Observed by Dr. T. A. McBride.—A male, aged twenty-eight years, seen at the New York Hospital in March, 1878. Complains of right supra-orbital neuralgia, which has lasted three months. The pain was constant at first, but latterly it has been paroxysmal and very severe. In the past week paresis of the right third nerve has supervened; patient has ptosis, dilatation of pupil, and external strabismus. On March 3d is ordered .0006 Duquesnel's aconitia in solution three times a day. Contrary to positive directions, the man did not report to Dr. McBride for four days, and then stated that he had been almost entirely relieved of pain; he had taken the medicine as directed until the evening of April 1st, when he stopped because of relief, and of tingling in tongue and ends of fingers. No change in paresis of motor oculi. Ordered sulphate of strychnia .003 t. i. d. The patient came regularly to the hospital for two weeks, and during that period there was no recurrence of pain.

CASE II.—Male; seen at the Manhattan Hospital by Dr. Seguin. An extreme case of epileptiform-trigeminal neuralgia, of two or three years' standing. In 1877 had derived great relief from Thompson's solution of phosphorus in full doses. Chief seat of pain is in supra-orbital branches of trigeminus, but all of its filaments in the left face sympathize in the attack. Was given .0006 of Duquesnel's aconitia twice a day for several days, with effect of provoking severe tingling, but without relief to pain or reduction in frequency of seizures. Patient not traced.

CASE III.—Male, aged about thirty-five years. Seen at clinic for diseases of the nervous system, College of Physicians and Surgeons, February, 1877. Old neuralgia of right infra-orbital nerves; epileptiform in type. Aconitia in doses of .0006 two or three times a day gave only slight relief; not enough to encourage continued treatment. Patient not traced.

CASE IV.—The reporter himself, in March, 1878, while weak from a combination of causes, had trigeminal neuralgia, involving all branches of the nerve on the left side, lasting six days. After failure of Thompson's solution of phosphorus, I tried aconitia, and took only .0003. Two doses were taken, with severe physiological effects. I felt much tingling in the fingers, legs, and tongue, had rigors, and was cold and faint. The only good effect was very slight and transient relief from severe pain. The attack was brought to a close by the extraction of a bad tooth in the upper jaw of the affected side.

CASE V.—Reported by Dr. N. B. Emerson at a meeting of the committee, held April 27, 1878.—J. D., aged thirty-two years, printer, presented himself February 15, 1878, suffering with attacks of violent pain in the first and second divisions of the right trigeminus, accompanied by clonic spasm of the facial muscles attached to the angle of the mouth on the same side. The pain was lightning-like in the suddenness of its onset, and was of the most acute form, causing him at the time of the attack to writhe with agony, and press his hands against the painful cheek. The affected side of the face was extremely sensitive. The paroxysms were very frequent. He had been similarly affected eight months before, and successfully treated by me with phosphorus and cod-liver oil. Present attack has not lasted long. No syphilis. There were several decayed teeth in the jaw, but they were not sensitive, and, in my opinion, were not likely to be the cause of the affection. Quinine, phosphorus and cod-liver oil, and morphine, were unsuccessfully used. I then decided to use aconitia, after Gubler's plan, and ordered :

℞.	Aconitiæ cryst.,	.01
	Spts. vini rect.,	q. s.
	Aquæ, q. s.	ad 62.—
	M.	

The first preparation was used two days without effect. I then directed the patient to have the prescription filled by Mr. Neergaard. At once .0006 produced entire relief of pain, followed by numbness of the mouth, tongue, and face, with peculiar symptoms in the periphery. On the recurrence of pain the following day, .001 was taken with less physiological effect, and less relief. On the third day, two doses of .001 each were taken night and morning, the terrible pain being relieved only after the second dose. Finally, after a dose of .0013 the pain remained entirely absent for eight days, and then returned with severity.

Case VI.—Observed by Dr. Seguin at the College of Physicians and Surgeons.—Mrs. A. D., aged fifty-seven; was first seen at clinic for diseases of the nervous system in the autumn of 1874. She gave the following history. In 1870 had trouble with the teeth in the right lower jaw, "caught cold in the gums," and present pain began. It occurred in paroxysms of sharp, severe pains in the right lower jaw, right half of tongue, and right half of lower lip. She suffered with no intermission up to the time when Dr. D. M. Stimson sent her to the college. The medicinal treatment which I then advised had no more effect on the neuralgia than other modes which had been tried, including extraction of the teeth.

In the succeeding summer, 1875, Mrs. D. again came to see me, representing herself as under no physician's care. I accordingly took charge of her, and excised at least 6. mm. of her inframaxillary nerve, by the intra-buccal method, also known as Lizars's. This was followed by absolute cessation of all pain in lip, tongue, and jaw, and by anæsthesia of the right half of the lower lip.

In a few weeks, patient thinks three or four, some return of sensibility occurred in the anæsthetic district, and has increased until now even delicate tests reveal no anæsthesia. No pain recurred until the early spring of 1877, a period of twenty months. In April, 1877, patient's husband died, and she sat a long time near the ice-box in which his body was preserved. Immediately had a return of neuralgic pain in the same regions, viz., tongue, gum, and lower lip of right side. The pain was again sharp and paroxysmal. She suffered greatly until late in the autumn of 1877, when spontaneous relief took place, and she had pain only at intervals during the whole winter. The only medicine which she took during this time was cod-liver oil. She had no powerful drugs. In the spring and early summer of this year she had as frequent and as severe attacks of pain as at any time; many paroxysms each day, attacks epileptiform in suddenness of appearance and in severity. She presented herself at the clinic for diseases of the nervous system for the third time, on July 13, 1878, and the following notes from the clinic case-book embrace her history since that date :

July 15*th*.—The pain begins in the gum of the right lower jaw, then darts into the right half of tongue along its whole length, especially in its anterior portion; it also affects the right half of the lower lip. She has no pain in the upper jaw or in the distribution of first branch of trigeminus, but it should be stated that she has a good deal of pain, also neuralgic in character, in the right side of the head behind the ear, the right side of the neck, and right shoulder. From almost the commencement of her illness, more or less of this pain has existed, varying greatly at times, but not annoying so much by far as the maxillary neuralgia. The paroxysms of pain in the jaw and tongue came on every few minutes. Once in a while, the patient adds, when the pain is greatest in the above-described region, a little of it shows itself in the gum of the right upper jaw. Is ordered a tonic mixture.

July 20*th*.—Is better, generally, than last week. Ordered extract gelsemini fld., five drops t. i. d., the dose to be increased by one drop each day.

July 27*th*.—Pain relieved by the gelseminum, seven drops of which produced queer sensations and double vision. In the last few days has taken only six drops t. i. d. Ordered five drops twice a day and ten drops at bedtime.

August 3*d*.—No marked benefit from above treatment, although much distress was produced by the doses taken. Ordered .00045 of Duquesnel's aconitia in solution t. i. d.

August 10*th*.—On the 7th reported at my office, and as the above doses had produced no effect I directed her to take .0006 t. i. d. on an empty stomach. To-day (three days after beginning the larger doses) she is free from neuralgic pain, though some soreness of the parts remains. After each dose of .0006 had some tingling in extremities and face. Treatment to be continued.

August 31*st*.—Has had no paroxysm of pain since beginning the .0006 dose. Has only noticed an occasional soreness in the tongue, provoked especially by acids. Can eat with comfort, whereas three weeks ago attempts at mastication caused agony. States that effects of one dose of aconitia consist in tingling in the whole body, most marked in the toes and fingers, and in peculiar chilly sensations.

The pain in the neck and shoulders is not wholly relieved. Complains of much sweating at night. To take for two or three days one .60 dose of sulphate of quinia at bedtime. The aconitia to be omitted, and Fowler's solution to be taken instead, in doses of three drops after meals, gradually increased.

September 14*th*.—Has remained perfectly free from facial neuralgia, and has only moderate pain in side of neck, right shoulder, and upper arm. Has taken ten drops of Fowler's solution without unpleasant effects; sweating arrested. Ordered to cease taking arsenic, and to use 4.cc. of Thompson's solution of phosphorus (=.003 of phosphorus) night and morning.

September 21*st*.—Had slight return of pain in right lower jaw and tongue on September 18th and 19th, arrested by a few doses of aconitia. To-day is perfectly well, except that right side of neck and arm is painful.

October 11*th*.—Has had no return of neuralgia since last note, and neck has not been so painful. States that she has more or less pain in the whole right side, from behind the ear to arms and down lower extremity to heel at times. With exception of slight neuralgic pains on September 18 and 19, has had no recurrence of inferior maxillary or lingual neuralgia since August 7th, a period of sixty-five days.

I append a case of another form of pain, viz., the severest fulgurating pains of sclerosis of the posterior columns, in illustration of the toleration of large doses of aconitia.

CASE VII.—Mr. B.——, 57 years of age, has suffered from typical fulgurating pains in the lower extremities for twenty-seven years. He has as yet no trace of ataxia, and, the only other symptom of spinal disease present is mydriasis of the right eye.

One of the favorite seats of these pains has been the internal aspect of the right leg, and in the last six weeks Mr. B—— has had innumerable paroxysms of cutting, tearing, and grinding pain in this region, sometimes causing extreme agony. Wishing to try aconitia for the relief of these pains, I gave him at first .00045 three times a day, and, not obtaining any relief or any physiological effects, gradually increased the dose to such a point that in forty-eight hours, ending October 4th, he consumed .01 without relief to pain, and with no physiological effect, except a transient and doubtful tingling in the finger-tips. I did not care to push the remedy farther. I should add that the prescription was filled at Neergaard's, and that I took pains to make inquiries as to possible errors in its preparation.

From the above cases the following conclusions may be justly drawn, I think:

1. The susceptibility of individuals to Duquesnel's aconitia varies enormously; one individual in the series having been severely affected by .0003, while another tolerated with no special symptoms .0008 every three hours. On the average, distinct physiological and therapeutical effects were obtained by giving .0006 three times a day.

2. Out of six cases of severe trigeminal neuralgia, one, probably a reflex neuralgia from a decayed tooth, was not at all benefited.

Three cases, epileptiform in form, were slightly or only temporarily relieved. Two cases were cured. One of these had existed for seven years, with an interruption of twenty months, procured by resection of the affected nerve.

It would thus appear that while we cannot indorse Prof. Gubler's statement that Duquesnel's aconitia never fails, we must recognize in it one of the most powerful and best agents for relieving and curing trigeminal neuralgia.

3. We do not as yet know the forms of trigeminal neuralgia which can be most influenced by aconitia. The three following cases have been reported to the committee since meeting of the society at which this report was read.

Case VIII. Observed by Dr. O. B. Douglas.—Mrs. C. H. M., aged nineteen, born in New York; married; was, October 19th, attacked with severe neuralgic pain in left eye—extending to submaxillary and bregmatic regions—which continued increasing in severity for three days and nights, being much worse at night, till she could only walk the floor and cry from pain. On the fourth day I saw her, and ordered, commencing at 10 A.M., drop doses of tincture of aconite root, beginning with four doses the first hour, two the second, and one each subsequent hour till relieved or physiological effects produced. The pain subsided, and she slept well the following night till 2 A.M., when, with slight return of pain, she awoke, took two doses, and slept till morning, and has had no return of the trouble to this date (Oct. 25th).

Last winter she suffered two weeks from a similar attack, and has been subject to neuralgic pains at other times, usually a result of exposure to cold. In all fourteen doses were taken, but no physiological effects of the drug were observable.

Case IX. Observed by Dr. A. H. Smith.—Mrs. R., aged forty-four years; married; seen October 13th. Had been suffering with severe pain in the face for four weeks, pain beginning in the right side, then passing to the left temporal and frontal region, and also affecting the left arm. It was aggravated to such an extent by the recumbent posture that the patient was unable to lie down. The night of the 12th was passed in extreme pain. Ordered the .00045 of Duquesnel's aconitia to be taken every four hours. Two doses were taken on the 13th with a slight degree of relief. On the 14th ordered the medicine to be taken every three hours. There was a decided abatement of the pain. At seven o'clock in the evening the patient experienced a numb and tingling sensation in the lips and tongue, and more or less over the whole right side, and especially in the fingers of the right hand. *It was not felt at all in the parts affected by the neuralgia.* The sensation was so decided that the patient, although forewarned, was considerably alarmed.

The night of the 14th was passed very comfortably, as was also the following day. During the evening of the 15th, however, the pain returned with great severity, but in the right instead of the left side. The medicine had been taken every three hours and a half; directed it to be taken at intervals of three hours. On the 16th there were again decided numbness and tingling, affecting this time chiefly the *left* side, the pain being chiefly in the right. The pain was greatly mitigated during the day, but returned every evening between seven and eight o'clock, lasting three to four hours. The 19th and 20th, however, passed by without a paroxysm. After that there was a recurrence every alternate day at about 5 P.M., lasting four hours. On the 21st quinine was ordered; the aconitia continued. Did not see her again until to-day (26th). Quinine had produced nausea, and had not been efficiently taken. Paroxysms have continued regularly and with unabated severity. Physiological effects of aconitia limited to tingling in tongue and lips. During one day the dose was repeated every two hours.

Case X. Observed by Dr. Seguin.—Male, aged thirty-nine. Epileptiform neuralgia on right side, involving all branches except lingual, for ten years. Suffering atrocious; many paroxysms a day. The case is under treatment, and is not ready for report, but I may say that Duquesnel's aconitia, given in doses of

.0006 three and four times a day, has produced physiological effects and diminished the severity of the disease. In the last week patient has had only one or two severe paroxysms a day, and few slight pains. The relief is so great that patient uses extravagant expressions of gratitude, "is in heaven," etc. This is the first treatment which has relieved him. At this date (October 27th) he is still under treatment, taking .0006 three times a day; iodide of potassium (no syphilis), and dialyzed iron.

These cases do not alter, but only confirm the committee's conclusions, as expressed *supra.*

A CONTRIBUTION TO THE MEDICINAL TREATMENT OF CHRONIC TRIGEMINAL NEURALGIA.*

HAVING recently met with three cases of severe chronic neuralgia of the trigeminus which have been favorably influenced by the internal administration of medicines, I have requested the privilege of presenting a report upon them to the Society.

CASE I.—*Epileptiform neuralgia of thirteen years' standing: cure.*—J. W., a farmer, aged 63 years, presented himself at my clinic for Diseases of the Nervous System on or about June 15, 1878, and gave the following history: Has suffered from neuralgia in the right side of the face for thirteen years. The first pain, slight and stinging, made its appearance near the external angular process of the frontal bone. There was a gradual increase in the frequency of the paroxysms, and in the severity of the pain, until the time of examination. During three years has had almost constant pain, *i.e.*, the paroxysms have been repeated every two or three minutes. There has been much pain at night, but the greatest suffering has always been experienced in the forenoon. The seat of neuralgia has been the right malar region and the lower anterior temporal region. Paroxysms have been excited by the contact of clothing or of the finger; by talking or eating, and by pulling the hair on the lip and cheek. The pain has never been periodical.

The patient's general health has always been good; he has had two attacks of malarial fever: one when a boy, the last six years ago. When the attack began he was living in Marlboro, Ulster Co., N. Y., considered a healthy place. Has never had syphilis; has always been temperate.

Attack witnessed at the clinic: A sharp and exceedingly severe pain appears in the region defined above, accompanied by injection of the cheek and eye, and the escape of tears. The paroxysm lasts several seconds, and returns every two or three minutes. Nitrite of amyl seems to mitigate the suffering. Examination of the affected and of adjacent parts is negative; there are no anæsthesia or true tender points, or any exciting cause of pain within the mouth. The etiology of the affection is unknown.

Treatment.—From June 17th to 21st, hypodermic injections of Squibb's chloroform were made daily through the mucous membrane of the cheek toward the malar region, from one to ten minims being used each time. In making these injections care was taken to avoid the point of exit of the infra-orbital nerve. The last injection was made near the supra-orbital nerve. These injections produced some smarting pain and secured relief for several hours each day, but did no more; the pain returning the next day as severely as before. Some bad effects were, however, produced, and these are worthy of consideration because hypodermic injections of chloroform in the face are usually considered harmless. I observed in this case some swelling at the seat of injection, paresis of the lower facial muscles of the type produced by lesions of the cerebral hemispheres; there was also marked numbness and slight anæsthesia in the skin of the cheek near the angle of the mouth, and over the eyebrow. The electro-muscular reactions remained normal, no abscess followed, and the paresis gradually passed away. I might add that similar unpleasant results ensued in another case in my practice about a year ago.

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* Read before the New York Neurological Society, Dec. 2, 1878. Reprinted from the N. Y. *Medical Record*, Jan. 4, 1879, Vol. XV.

On June 26th, 27th, 28th, daily injections of Fowler's solution (diluted one-half) were made in the affected cheek through the mucous membrane without good or bad effects.

From June 21st to 26th, I tried Thompson's solution of phosphorus, in doses of one teaspoonful (.003 of phosphorus) three and four times a day without marked benefit.

Still, on the whole, at the end of June, the patient was somewhat improved, having severe paroxysms only from four to ten times a day; though slight, sharp pains were still very frequent.

About the end of June he was given iodide of potassium in gradually increasing doses of a saturated solution. He began with ten drops three times a day, and by an increase of five drops per day at each dose, he attained a maximum of ninety-five drops three times a day. No evident benefit resulted from this course, which was terminated on July 12th.

On July 13th, was ordered five drops of the fluid extract of gelseminum four times a day. July 15th.—Reports himself as very much relieved; no special symptoms have been produced by the drug; is directed to take eight drops four times daily. July 16th.—Yesterday had no paroxysm except while eating; there have been frequent but bearable "ticks" of pain in the vicinity of the right external angular process of the frontal bone. Is ordered to take ten drops four times a day.

August 1st.—About this time, as the patient could no longer stay in town, and as I was unwilling to let him take glesemiuum while away from observation, the solution of iodide of potassium was again given in doses of sixty drops three times a day.

August 10th.—Patient returns to town, and reports himself no better; he has taken the medicine regularly, and has kept a journal of the attacks. The number of attacks per diem, usually excited by eating, etc., have varied from four to eight. The iodide is suspended. The actual platinum cautery is gently applied over the right malar and temporal regions, and five drops of Fowler's solution are given in water three times a day, to be gradually increased. August 20th, the diary shows a decrease in the number and in the severity of the pains; only from three to five paroxysms a day; three yesterday. Has been cauterized three times.

August 22d.—About this time the neuralgia ceased altogether, the dose of Fowler's solution being ten drops three times daily.

September 22d.—Patient has had no pain since the last note—a period of thirty-two days. Absolutely no pain has been felt, and the hyperæsthesia has disappeared; patient can eat, talk, wash, or rub his face with impunity for the first time in many years. The paresis of the lower face, produced by the injections of chloroform, has nearly passed away, and there is no more numbness. No toxic effects have been caused by the arsenic; but, as he has taken ten drops so long, a change is made to Thompson's solution of phosphorus, one teaspoonful three times a day.

On September 24th a few slight paroxysms occurred, and the patient, of his own accord, resumed the arsenical solution in full doses, and in a day or two the pains ceased, and they have not returned.

Early in November this patient was shown at my clinic. He then asserted that he was perfectly well, and his healthy and cheerful aspect confirmed his statement. As he has not returned, I feel reasonably sure that the good result has been permanent.*

CASE II.—*Epileptiform trigeminal neuralgia of ten years standing greatly relieved by treatment.*—H. S., aged 20 years, a janitor by occupation, consulted me on October 2, 1878, and gave the following history: Syphilis was denied. Previous to the development of the present affection he had been subject to occasional dull headaches. Ten years ago he suddenly experienced a very severe sharp pain all through his head, "as if devils were at work there," lasting half an hour. There was no dizziness, or nausea, or faintness, or impairment of sight, or paralysis. For a period of six months he remained free from pain, and, indeed, was perfectly well; then a "dull, stupid pain" began over the right eye, extending from the supra-orbital notch inward to the nose, and down the side of the nose to the ala. This pain was paroxysmal, and worse in the day-time. Later the pain extended to the eyeball, and was exceedingly severe; the paroxysms recurring from ten to twelve times a day. In the course of two or three years, pain made its appearance in the right temple, worse at night.

In the last few years the most pain has been on the top of the head, above the temple, and in front of the ear to the bregma. There has lately been an occasional and rare pain in the nose ; not much in the temple. During the past summer and since, there has been some occipital pain on both sides, more on the right. In the last year there has also been pain in both jaws, in the upper lip near the median line ; none in the tongue. In the last four years vision has been dim, and glasses have not corrected this defect. Five years ago, while taking medicine, had temporary diplopia. At various times during this long illness has had "dizzy spells" with varying frequency ; seldom in the last few months. Has had no symptoms in other parts of the body ; memory is impaired ; the virile power quite lost. Had severe dyspepsia and vomiting three years ago, and has been costive during the whole period of the disease. The various painful regions are hyperæsthetic, but not numb, and the tactile sensibility is perfectly preserved on both sides. There is no facial paralysis ; the right pupil is positively small, the left normal. After dilatation by atropine, the ophthalmoscope shows nothing abnormal in the bottom of the eye. Hearing, smell, and taste are normal. The urine has been frequently examined by physicians and always found normal ; it is now free from albumen. Marked anæmia is present in the skin and mucous membranes ; has always been pale.

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* A letter from this patient's wife, received about December 10th, states that he remains well.

The paroxysms of pain are the most terrible which I have ever witnessed ; the patient fairly writhing in his chair or falling to the floor in his agony. During the attack the right eye is very much injected and waters.

The patient states that no medicine has ever relieved him, and he has tried a great many. I at once prescribed Duquesnel's crystallized aconitia, a remedy with which I had obtained remarkable results during the year. The prescription was :

R. Aconitiæ (Duquesnel's)	.008
Spts. vini rect.,	
Glycerinæ, āā	4.cc.
Aq. menthæ pip. ad	62.cc.
M.	

S.—A teaspoonful three times a day between meals.

I also gave him one teaspoonful of Wyeth's dialyzed iron every evening at bed-time.

Oct. 3d.—Has severe paroxysms every day ; seven on October 3d, and nine yesterday.

Oct. 11th.—Has only slight physiological effects (numbness) in the finger-tips ; from six to nine attacks each day. Now takes .0008 aconitiæ three times a day.

Oct. 14th.—On the 12th had twelve severe spells ; only two yesterday. He yesterday took, by mistake, 8.cc of aconitia solution, or .0015, twice, and two doses of 4.cc, and this morning 8.cc. This is the equivalent of .006 of aconitia in twenty-four hours. He is very nervous, feels as if electricity were passing through his body and limbs ; he "cannot contain himself." As this was a mistake, I directed him to resume the prescribed doses of 6.cc ter die. The results of the mistake were, however, most fortunate ; improvement began from this strong impression of aconitia upon the system, as shown in the tabular record of paroxysms :

Oct. 19th.—Excellent record ; since October 13th has had only from one to three severe attacks ; ordered to continue aconitia and to begin a saturated solution of iodide of potassium in five drop doses.

Oct. 31st.—Continues to do well, *i.e.*, has from one to two or three severe paroxysms daily, and a number of slight twinges. Feels numb and "very cold" from three doses of aconitia. Can't be warmed even by an overcoat; general condition much improved ; physiognomy calm and contented. Besides aconitia, takes twenty-eight drops of solution of potash.

Nov. 30th.—Improvement maintained. Passes some days without severe attacks, and a few with no pain at all. Has done much of his work as janitor of late. The aconitia has lately (since 23d) been taken twice a day, and he has hardly any numbness.

On Dec. 19.—Pills of arsenic .004, quinia .20, and belladonna, .02, were substituted for the iodide of potassium. The iron is kept up at night, 4.cc. of dialyzed iron.

CASE III. See p. 370.

It seems to me that three conclusions may legitimately be drawn from the above related cases:

1. That there is a possibility of relief in most severe cases of epileptiform trigeminal neuralgia. The usually received opinion is that, in such cases, recourse must be had to operation upon deep branches of the nerve, excision of Meckel's ganglion, etc., and to the systematic use of morphia to make life endurable. After my experience with the above cases, I am disposed to urge a sufferer from trigeminal neuralgia to make a trial of medicinal treatment.

2. The advantage of using medicines systematically. Not only should the doses of any one remedy be administered regularly and in progressively increasing doses, but several remedies may be used in succession, so as to profoundly affect the system. Of the medicines applicable for the treatment of neuralgia, the following are those which I can recommend most highly: aconitia, arsenic, iodide of potassium, gelseminum, belladonna, quinia, morphia, galvanism, the actual cautery, Thompson's solution of phosphorus.

3. In the treatment of chronic neuralgia and of many neuroses, it is necessary to obtain the physiological effects of the drug employed, in order to do good. This principle of heroic medication is one which ensures success in seemingly desperate cases, and its execution requires the utmost watchfulness on the part of the physician, and intelligence and faithfulness on the part of the patient and his attendants. Many unpleasant consequences of such treatment may be avoided if we at first give very small doses of the remedy, and then make a very progressive increase. The good effects of giving medicines to the production of physiological effects are illustrated in the above cases; in the treatment of chorea by arsenic; of malarial affections by quinia; of spinal congestion and myelitis by belladonna; of syphilitic disease by mercury and iodide of potassium, etc., etc.

Inasmuch as the good effects noted in cases II. and III. were obtained by the action of Duquesnel's aconitia, it may not be amiss to refer here to the conclusions of a report on aconitia recently made to the New York Therapeutical Society by its Committee on Neurotics. *

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* *Vide*, p. 367, also N. Y. *Medical Journal*, 1878, p. 621, Vol. 28.

DERMATITIS PRODUCED BY THREE PREPARATIONS OF OPIUM IN THE SAME SUBJECT.*

The following case, illustrating the unexpected results which may follow the moderate use of well known remedies, was of great interest to the Committee on Neurotics of the N. Y. Therapeutical Society, and is perhaps worthy of record. The patient was a lady about fifty years of age, of nervous temperament, and of generally good health. When I first saw her on September 24th, 1878, she had been suffering for several months from mild melancholia; her chief symptoms being sadness, delusions as to her wickedness, hopelessness of recovery through human agency, and insommia. On my proposing the opium treatment which is my main reliance in such cases, I was told that some time ago a very small dose of morphine had produced an erysipelatous eruption. Unwilling to give up my mainstay in treatment, I preferred to run the risk and prescribed a pill composed of extract of opium, .008 extract of cannabis indica, .015 and quinine, .12, to be taken three times a day. The next morning, after having taken three pills, Miss L. called and showed me upon her neck an erythematous patch almost encircling it, and which was the seat of extreme burning and itching. She stated that several such patches were on other parts of the body. The patches appeared red and felt hot; they presented neither papules or vesicles. This eruption exactly resembled, the patient said, the "erysipelas," which had on several occasions been caused by morphine and opium. It should be added that although the patient had no knowledge of my prescription, she positively expressed her belief that I had given her opium. On the third day the symptoms were aggravated. In addition to erythema of the neck, there was a distinctly erysipelatous condition of one upper eyelid; characterized by redness, œdema and tenderness. The opium was now (Sept. 26th,) discontinued, and in a few days the skin resumed its usual healthy appearance.

The patient steadily improved in her mental condition, under the use of dilute nitro-muriatic acid and strychnia before meals, and of camphor and cannabis pills at bed-time.

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* From the *Archives of Medicine*, Feb., 1879.

On November 11th, I ordered in place of the above pills, others composed of cannabis, .06, camphor, .12, codeia, .03, aloes, .03; one to be taken at night. On the very next morning, (Nov. 12,) the unfortunate lady exhibited an erythma on neck, shoulders and body, the left eyelid was red and a little swollen. She was told to resume pills without codeia, and very soon all trace of the cutaneous affection disappeared.

MYELITIS OF THE ANTERIOR HORNS OF TRAUMATIC ORIGIN.*

F. M., a carpenter, twenty-seven years of age, presented himself at my clinic for Diseases of the Nervous System, on September 20th, 1878, and gave the following history. On the sixth of April last he fell from the fifth story of a building, and dropped obliquely upon the sidewalk and curbstone, striking his hip and side. He did not lose consciousness, and there was no marked external injury. He immediately experienced partial paralysis of both lower extremities, and of the left upper extremity. In four weeks recovered the use of his arm. In six weeks later he began to improve in his lower limbs, and by July 20th, could walk without crutches. At no time was there any loss of sensation, though there was a degree of numbness and tingling of the paralyzed parts. Had no pains, cramps, or jerking of these parts, but he well describes epileptoid trepidation. Urine was retained (catheter used) for three weeks after accident, rectum unaffected; never incontinence of urine. Since July has regained his normal state except a peculiar disability in his legs, and burning in the toes of both feet, which perspire freely.

Examination: Patient free from symptoms above knees; below knees has atrophic paralysis of the anterior tibial group of muscles on both sides. In walking, the feet are brought to the ground in a slapping or flapping manner. Posterior tibial and peroneal muscles normal. No tendon-reflex at knees, but foot-phenomenon can be slightly produced on both sides. Sensibility normal. Electrical reactions; no faradic or galvanic reaction in

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* From the *Archives of Medicine*, Feb., 1879.

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anterior tibial nerve; no faradic reaction in anterior tibial muscles; galvanism produces slow contractions in the paralyzed muscles with the formula Ka CC = An CC. Up to present time (January 10th, 1879), M. has been treated with galvanism to the legs, ergot and iodide of potassium. Although several accidental causes have interrupted the treatment, marked improvement has taken place. Some voluntary power has returned in all the muscles of the left anterior tibial group, and in the tibialis anticus of the right side.

It would seem as if a sudden hyperæmia of the cord had been produced by the fall, with hemorrhage or myelitis in the lumbar region limited to a small part of the anterior horns, seriously injuring those ganglion cells which give origin to the nerve fibres innervating the anterior tibial regions. One case of traumatic infantile paralysis (lesion of the anterior horns), by Dr. Allbutt, (*Lancet*, 1870, II., p. 84), and another in the adult by Prof. Leyden, (*Archiv für Psychiatrie und Nervenkrankheiten*, vi. p. 271), have been placed on record. A number of cases of traumatic spinal paralysis in the course of which muscular atrophy appeared, are also recorded by Ollivier, Gull, and Erichsen.

THE PRESENT ASPECT OF THE QUESTION OF TETANOID PARAPLEGIA.[*]

SYNONYMS, *Spastische Spinalparalyse*, ERB; *Tabes dorsal spasmodique*, CHARCOT.

IN the last five years a new symptom-group, indicating disease of the spinal cord has been independently observed and described by several physicians.

In 1873 I published under the somewhat unfortunate name of "tetanoid pseudo-paraplegia," five peculiar cases which I had studied in the three preceding years.[†] I then defined this "peculiar paraplegiform affection" as follows: "This form of false paraplegia (using the word as implying the existence of paresis or akinesis in the lower limbs) is characterized by impairment of the functions of the lower extremities, when the patient is in the erect posture, without any loss of power in these parts. Further analysis shows that the seeming paraplegia is dependent upon tonic spasm of the muscles of the lower limbs. As negative characters we have absence of the symptom ataxia, and often, also, preservation of sensibility."

Possibly I may be pardoned for claiming that this paragraph contains, with some errors, the essence of the symtomatology of the condition now under consideration.

In May, 1875, Prof. W. Erb, of Heidelberg, who had seen my essay, read a paper, before the Association of Physicians for Nervous and Mental Diseases held at Heppenheim,[‡] upon a condition of spasmodic spinal paralysis, in which he describes cases of paresis of the lower limb complicated with stiffness and even contracture of the lower limbs, without anæsthesia. In this paper he gave a description of the "spastic walk," in terms not very different from those I used in 1873; substantially the same essay was reproduced shortly afterward in the *Berliner*

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[*] Reprinted from *Archives of Medicine*, February, 1879.

[†] Description of a peculiar paraplegiform affection. See Vol. I., p 127; also *Archives of Scientific and Practical Medicine*, February, 1873, New York, p. 101.

[‡] W. Erb. Ueber einen wenig bekannten spinalen Symptomencomplex. *Zeitschrift für Psychiatrie*, Band XXXII., 1875. Also in *Berliner klinische Wochenschrift*, 1875, Band 12, p. 357.

Klinische Wochenschrift, and since, the condition of spastic spinal paralysis has had a place in nosology. It has not been noticed in the various articles upon tetanoid paraplegia that, in his Treatise on the Diseases of the Nervous System[*] (preface dated March 10th, 1876), Prof. Hammond described this rigid paralysis, and referred to Türck's and Charcot's pathological observations in cases of sclerosis of the lateral columns. He accepts the symptom-group as a natural one.

In 1876, Prof. Charcot[†] of Paris, who had observed the symptoms before becoming acquainted with Erb's work, delivered one or more clinical lectures upon the subject, calling the affection *Tabes dorsal spasmodique.*

In the same year Charcot's views were reiterated by one of his pupils, J. Bétous, in his inaugural dissertation.[‡]

In both these publications the opinion is expressed that the symptoms which I prefer to designate as tetanoid paraplegia, are caused by primary disease of the lateral columns of the spinal cord, a form of sclerosis, either of the disseminated or of the systematic type.

This proposition of Charcot's was based upon an autopsy in a case of so-called hysterical contracture, in which he had found[§] symmetrical sclerosis of the lateral columns, and upon the results of two autopsies by Ludwig Türck[‖] in 1856. Unfortunately, the notes of these cases are not of such a nature as to afford us any aid in the study of the symptom-group under consideration (Erb).

In 1877, Prof. Erb wrote a more elaborate essay[¶] upon the symptom-group in question, which he then designated by the term spastic spinal paralysis, and which he, following Charcot, believed to be due to primary sclerosis of the lateral columns of the spinal cord. More recently still, in Vol. XIII. of the American edition of Ziemssen's Cyclopædia, he has reiterated his views unchanged.

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[*] A treatise on the diseases of the nervous system. Sixth Edition. New York, 1876, p. 569.

[†] Leçons sur les maladies du système nerveux. T. II., 15me Leçon, p. 275, 2me Edition, Paris, 1877.

[‡] Étude sur le tabes dorsal spasmodique. *Thèse de Paris*, 1876.

[§] *Gazette Hebdomadaire*, 1865, p. 109.

[‖] Ueber primare Degeneration einzelner Rückenmarksstränge. *Sitzungsbericht der k. k. Akademie zu Wien*, 1856.

[¶] Ueber die spastiche Spinalparalyse (Tabes dorsal spasmodique, Charcot). *Virchow's Archiv für pathologishe Anatomie*. Band LXX., 1877. Idem, *Ziemssen's Cyclopædia of Practice of Medicine*, American Edition, Vol. XIII., p. 620.

Since Erb's and Charcot's first publications, numerous contributions to the literature of the subject have appeared, among which I may name O. Berger,[*] F. Richter,[†] Seeligmüller,[‡] R. Schultz,[§] M. Rosenthal,[‖] Stofella,[¶] and L. C. Gray (of Brooklyn).[**]

These writers have, with little or no qualification, accepted the Erb-Charcot views of tetanoid paraplegia.

Two eminent physicians have, however, protested against these views as too exclusive, as tending to elevate the symptom-group to the rank of a well-defined disease. These are Profs. Westphal[††] and Leyden.[‡‡] Prof. Westphal relates in the *Charité-Annalen* an extremely interesting case of paraplegia with rigidity, a little diminution of sensibility and marked numbness in the lower extremities, without cystic or rectal paralysis. This patient had recovered almost perfectly at the date of writing the report (July, 1877). Assuming that the symptom-group in this case was similar to the spastic paralysis of Erb, Dr. Westphal proceeds to state his belief that paraplegia with rigidity may be produced by a variety of spinal lesions; especially, in his experience, by early and unrecognizable Pott's disease of the vertebræ. This last statement is in remarkable accord with the pathology of my own cases, three out of five having been of this nature.

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[*] O. Berger. Die primare Sklerose der Seitenstränge des Rückenmarks. *Deutsche Zeitschrift für praktische Medicin*, 1876, Nos. 16–19.

Idem. Zur lehre von der primären Lateralsklerose des Rückenmarks. *Deutsche Zeitschrift für praktische Medicin*, 1877, Nos. 3, 5, 6.

[†] F. Richter. Zur Sklerose der Seitenstränge des Rückenmarks. *Deutsches Archiv für klinische Medicin*, 1876, p. 365, Band xvii.

[‡] Seeligmüller. Sklerose der Seitenstränge des Rückenmarks bei verschiedener Kindern derselben Familie. *Deutsche Med. Wochenschrift*, 1876, Nos. 16, 17, Band ii., pp. 185–197.

[§] R. Schultz. Mehrere Fälle von "Lateralsklerose." *Archiv der Heilkunde*, 1877, xviii., p. 352.

[‖] M. Rosenthal. Traité clinique des maladies der système nerveux. Paris, 1878, p. 406.

[¶] Von Stofella. *Wiener Med. Wochenschrift*, 1878, Band xxviii, pp. 565–594. *London Medical Record*, June 15th, 1878, p. 272.

[**] L. C. Gray. Spasmodic spinal paralysis. Proceedings of the Medical Society of the County of Kings, 1878, No. 29, p. 167.

[††] C. Westphal. Allmälige enstandene Paraplegie mit Rigidität. *Charité-Annalen*, 1876, p. 372. Berlin, 1878.

[‡‡] E. Leyden. Ueber spastische Spinallähmung. *Berliner klinische Wochenschrift*, 1878. Band xviii., pp. 706, 725.

Prof. Leyden has very lately ably reviewed the subject in its various relations in a paper read to the Berlin Medico-Psychological Society.* In this communication, while admitting that sclerosis of the lateral columns may be primary, and that this lesion will probably cause rigidity of the limbs below the lesion, he maintains his former position, to the effect that "spastic paralysis" often is the expression of a chronic dorsal myelitis. Experience since 1875 has convinced him that many spinal lesions may cause "spastic paralysis," as (1) traumatic myelitis, (2) compression of the spinal cord by Pott's disease or by tumors, (3) spontaneous chronic myelitis (disseminated sclerosis involving the lateral columns), (4) spinal paralysis after acute disease, (5) syphilitic paralysis, and (6) spinal meningitis or peri-myelitis.

Leyden proposes a clinical subdivision of spinal paralysis into two classes, supple or atonic paralysis, and tonic or spastic paralysis.

The latter form (including the Erb-Charcot symptom-group) is explicable upon three hypotheses: (1) from increased excitability of motor nerves, (2) from increased excitability of the sensory roots of nerves, and (3) by partial or total interruption of the voluntary conduction from the brain to the periphery, with increased reflex power of the spinal gray substance. He lays the greatest stress upon the last-named explanation, which has a physiological basis in the experiments of Goltz (inhibitory action of the brain upon the spinal cord). I gave this explanation of the spasm in my cases in 1873. Another recent critic of the Erb-Charcot proposition, Dr. Ricklin † of Paris, has pointed out that a number of cases of so-called spastic paralysis, in the essays of Richter, Berger, Schultz, and even Erb, might be looked upon as irregular cases of myelitis. He even objects to Stofella's case with autopsy, because of the absence of microscopic examination, and of any statement as to the condition of the upper spinal cord, mesocephale, and brain. Westphal and Leyden suggest that many of the cases of spastic paralysis now recorded are cases of localized (dorsal) myelitis, with secondary degeneration of the lateral columns below the lesion. It has been impossible for me to read Berger's[*] and Seeligmüller's[*] contributions in the original, but with respect to Richter[*] I can say that his cases are much more like partial myelitis, or meningomyelitis, than like spastic paralysis. Especially is this true of his case No. 4, which he reports as almost cured by electricity and hydrotherapy. Schultz[*] in his report, prepared under Erb's supervision, relates two cases (out of four) which are confessedly not typical.

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* E. Leyden. *Klinik der Rückenmarkskrankheiten*, Band ii., p. 445. Berlin, 1875.

† Ricklin. De la paralysie spinale spasmodique ou tabes spasmodique. ***Gazette Médicale de Paris***, 1878, pp. 321, 345, 369.

Nothnagel,[†] recognizing the general characters of a spastic case occuring under his observation (1876), relates it under the title of *dorsal myelitis.*

A survey of the literature of tetanoid paraplegia would be incomplete without a consideration of a functional form of it, first described by Dr. L. A. Sayre, of New York, under the somewhat extraordinary name of spinal anæmia. His first paper was published in 1870,[‡] and a second fuller exposition of his views was presented to the New York Society of Neurology and Electrology, March 1st, 1875, and printed in the Transactions of the American Medical Association for 1875. In these publications Dr. Sayre has described a form of spastic paralysis occurring in children, characterized by spasm of the adductor muscles of the legs, and of some muscles of the hands and arms, by paresis, and by a degree of inco-ordination. In many cases apparent idiocy was present, the clitoris or penis in these little sufferers was found red, irritable, and touching it produced local excitement and strong spasm in the limbs and body of the patients. Excision of the clitoris, circumcision, or simply tearing off and turning back an adherent prepuce, is reported to have wrought magical improvement in all, and a cure in most of the subjects.

Dr. Sayre quotes (Transactions of American Medical Association, 1875) Dr. Barwell of London, in such a way as to lead one to suppose that the latter observer had met with cases of spastic paralysis dependent upon sexual irritation. A reference to Barwell's lecture[§] will show that he described *paresis* and *paralysis* in such cases, and not contracture, spasm or inco-ordination. Dr. Sabal, of Jacksonville, Florida, has met with cases like Dr. Sayre's, and has been successful in their treatment.

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[*] L. c.

[†] **Nothnagel.** Beobachtungen über Reflex-hemmung. *Archiv für Psychiatrie und Nervenkrankheiten,* Band vi., case ii., p. 336.

[‡] **L. A. Sayre.** On reflex paralysis produced by phymosis and adherent prepuce. Transactions of American Medical Association, 1870. Vol. xxi., p. 205.

Idem. Spinal anæmia with partial paralysis and want of co-ordination, from irritation of the genital organs. Transactions of American Medical Association, 1875. Vol. xxvi., p. 255.

[§] **Barwell.** Lectures on infantile paralysis. *Lancet,* 1872, Lecture iv., 1872, ii., p. 551.

While recording these statements as to the existence of a functional tetanoid paraplegia, it should not be forgotten that Dr. Eugene Dupuy,* now of San Francisco, has said, "Dr. Sayre has lately recorded some very interesting cases of contraction in young children which gave way entirely after the operation for phymosis. In some cases operated on by Dr. Sayre, the relief has not been of long duration, if I am to judge from what I have seen, as some of those young patients have been under my care since the operation performed by Dr. Sayre, and are as yet suffering as much as before from the same troubles." Even if we accept this statement of Dr. Dupuy, it seems probable that a functional spastic paralysis may exist. There is certainly nothing in physiology or in laws of morbid reflex actions to make such a condition impossible *a priori.* I have, however, never met with such a case either before or after an operation. As to Dr. Sayre's theory of spinal anæmia, it is hardly worthy of discussion, and I infer from the wording of his articles that he himself does not attach much importance to it.

To conclude this clinical study of tetanoid paraplegia, I would briefly state my experience since writing my paper in 1873.

In the first place, I believe the facts therein stated to have been well observed, though perhaps I may have overlooked paresis in some of my patients. In some of them, it is positively stated that while recumbent, their resistance strength at the knees was normal. Case III. was recognized as a case of amyotrophic lateral sclerosis (Charcot) not long afterwards, and a complete post-mortem examination, which I have recently finished, has proved the correctness of this diagnosis. My explanation of the genesis of spasm is yet, I believe, tenable, and the chief error I am willing to admit is having too hastily drawn the conclusion that tumors compressing the cord were the anatomical cause of the symptoms. A more critical examination of Case IV. might have saved me from this error, as it was evidently a case of syphilitic myelitis in the dorsal region.

Second, I have met with several cases of dorsal myelitis (in accord with Westphal, Nothnagel and Leyden), in which at a certain period well marked tetanoid paraplegia, or a spastic condition, set in. One of these was Case IV. of my essay. Another was a young man (treated in consultation with Dr. Charles McBurney in 1873-4), who recovered from a very severe attack of syphilitic paraplegia, and who for a long time suffered from stiffness and awkwardness of both lower extremities, with increased reflex. This mild tetanoid state gradually wore away, and the patient has now been for several years perfectly well. A third case was also one of severe syphilitic paraplegia, from dorsal myelitis, in which recovery progressed to a certain point, and remained stationary in spite of remedies; the remaining symptoms being marked anæsthesia, paresis, great rigidity of the legs, especially when patient stood and tried to walk with crutches. Then the legs and feet were held tightly together in adduction, and only a violent effort enabled the patient to take a small step. Various forms of increased reflex were observed in this patient. A fourth case now under observation is that of a lady twenty-eight years of age, who in 1874, after confinement, had a severe complex illness in which myelitis played a part, and was paraplegic for several months. Since has had weak and numbish legs, with abnormal reflexes, especially of bladder and rectum. Her walk is done in small steps, the legs being very rigid; no dragging of feet, no loss of equilibrium when standing with eyes closed; legs strong enough when tested in bed; knee and sole reflexes are very strong.

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* Eugene Dupuy. Pathology and treatment of reflex motor symptoms, paralysis, contractions, etc. *Journal of Nervous and Mental Disease*, April, 1877, p. 232.

Still another case is one of destructive central myelitis of the cervical enlargement, characterized by atrophic paralysis of the hands, forearms and one shoulder; absolute anæsthesia up to the middle of the arms, partial anæsthesia of shoulders and parts of neck; contraction of the left pupil, and tetanoid state up to the left lower extremity, without anæsthesia or atrophy. This tetanized leg is on the same side as the greatest atrophy and the contracted pupil.

In the third place, I have seen several children with stiffened, contractured, adducted, and inco-ordinate limbs (upper and lower) co-existent with defective cerebral development. In other cases the legs alone were stiffened and adducted, and there were no symptoms in the upper extremities or head, and no sexual irritation.

Dr. Erb* has recently described similar infantile cases, in which the legs alone were weak and tetanized, without alteration of sensibility. At the present time I have a fairly well-marked instance of the symptom-group in a child of six years attending at my clinic.

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* W. Erb. Ueber das vorkommen der "spastischen Spinallähmung" bei kleinen Kindern. *Memorabilien*, 1877, 12 Heft.

From a careful consideration of the above data, I think that the only safe conclusions to be drawn now are:

1. There is possibly a *disease* worthy of being called primary sclerosis of the lateral columns, and characterized by tetanoid paraplegia without anæsthesia, ataxia, atrophy, or affection of the bladder (Erb-Charcot view).

2. There is very certainly a tetanoid paraplegia indirectly produced by various lesions of the spinal cord, as pressure effects (Leyden and myself), syphilitic and non-syphilitic myelitis in the dorsal region (Leyden, Westphal, Nothnagel and myself), amyoatrophic lateral sclerosis (Charcot, Leyden and others), traumatic myelitis (Leyden), disseminated nodular sclerosis (Charcot—Case IV. of Bétous' essay). Besides, the wonderful resemblance between the one-sided phenomena of late contracture in hemiplegia of cerebral origin and tetanoid paraplegia must be borne in mind.

3. It is probable that a functional tetanoid paraplegia exists in children, caused by genital or other peripheral irritation (Sayre).

4. It is possible that tetanoid paraplegia cervicalis in young children may be due to defective cerebral development, and consequently agenesis of certain tracts in the cord. This is rendered probable by at least one case which has come under my observation, and by a consideration of the hemi-spasm which follows grave cerebral lesions, and which we designate late contracture.

If we now turn to pathological anatomy, we obtain instructive information.

1. In the "true" spastic paralysis, Charcot and Erb claim that there is a primary sclerosis of the lateral columns of the cord. The opinion is based upon five autopsies: three by Türck,* one by Charcot,† and a more recent one by von Stofella of Vienna.* The claims of these autopsies to the rank of demonstrations is contested by several, especially by Ricklin.* The objections are that Türck's and Charcot's examinations were made in cases whose symptomatology was not well recorded, or

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* L. C. † L. C. *Gaz. Hebd.*

was that of another disease (hysterical contracture—Charcot). The last case, even though the autopsy was witnessed by Prof. Klob, is unsatisfactory, because (1) no microscopical examination was made; (2) nothing is said of the state of the upper spinal cord, medulla, pons and brain, and (3) it is stated that the sclerosis (of the posterior part of the lateral columns) grew less and less in the cervical region.

2. In the case which Charcot and Bétous took for spastic paralysis, and which was found to have been disseminated sclerosis, numerous nodules were found in the lateral columns.

3. In the late contracture of hemiplegia, which produces symptoms so much resembling tetanoid paraplegia, we now know beyond question that there is degeneration of the posterior part of the lateral column—the crossed pyramidal column of Flechsig.

4. In the various diseases of the spinal cord which, in the experience of many observers besides myself, have been accompanied or followed by a tetanoid state, we have good reason to believe that there was secondary descending degeneration of the crossed pyramidal column. For example, in my case of cervical myelitis with stiff left leg, it can hardly be doubted that the left lateral column (crossed pyramidal column) has undergone a degree of degeneration.

From the above it appears almost certain that lesions of the crossed pyramidal columns have much to do with the development of tetanoid paraplegia, though we as yet lack a demonstration of the existence of a *primary* sclerosis of these parts.

A few months ago I should have been disposed to make the relation between sclerosis or degeneration of the lateral columns (crossed pyramidal column) and the tetanoid state of the limbs one of imperative causation, but since then, Dr. J. C. Shaw, of Brooklyn, has exhibited preparations of the spinal cord of a patient who had progressive muscular atrophy without a trace of stiffness of the limbs, and in the specimens there exists degeneration of the ganglionic bodies of the anterior horns, and well-marked sclerosis of both crossed pyramidal columns. This sclerosis of the lateral columns was at least as well marked as in my own case of amyotrophic lateral sclerosis, in which the contracture was extreme. This rather puzzling case of Dr. Shaw's is published in the January number of *Jewell's Journal of Nervous and Mental Disease*, of this year. Quite a number of cases of amyotrophic lateral sclerosis, in which sclerosis of the lateral columns was found without there having been any rigidity of the muscles, are on record. Chief among them are those by Duménil, and by Barth.

With the treatment and prognosis of tetanoid paraplegia, this review can have nothing to do. I would, however, remark that several cases have been cured, including one which was recently reported from Kussmaul's clinic at Strasburg, by Velden.*

The question of priority of description of the symptom-group tetanoid paraplegia, of first claiming that there is a paraplegiform affection chiefly characterized by spasm, is one of minor importance, and I am quite ready to submit my claim in the language of Prof. Leyden,† "*Weniger bekannt ist, dass auch Séguin die spastische Paralyse als eine besondere Form geschildert hat, unter dem etwas schwerfälligen Namen der Tetanoid Pseudo-Paraplegia.*"

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* Velden und Kussmaul. Fall von spastischen Spinalparalyse. Heilung. *Berliner klinische Wochenschrift*, 1878. Band 15, p. 563.

† L. C. Berlin. *Klin. Woch.*, 1878, pp. 706, 725.

THE USE OF THE ACTUAL CAUTERY IN MEDICINE.*

THE question, "Where can an account of the actual cautery be found?" is so often asked me by students and practitioners, that I am led to believe that a brief statement of the mode of using this powerful agent in medicine, and an estimate of what it may reasonably be expected to accomplish, may prove interesting.

First, as to the instruments and mode of application used in past times. A very incomplete study of the literature of the subject reveals the fact that cauterization was used by the oldest physicians, and that, with remarkable oscillations, it has disappeared and reappeared in medical and surgical practice in past centuries.

About the end of the last century the celebrated French surgeon, Percy, made an elaborate report upon the subject of cauterization in general, in which he criticised the forms of cautery and their mode of application. His favorable report gave a great impetus to the use of this remedial agent. Surgeons have used it more than physicians, and the latter have continued to employ blisters, cupping, etc., for purposes of counter-irritation.

Most various forms of cautery have been employed; olivar, crescentic, linear, etc.

Numerous materials have been employed to make the cautery; iron, silver, gold, platinum, each metal being supposed to possess special advantages. The introduction of the platinum cautery is generally attributed to Dr. Brown-Séquard; but I find that Hoppe, a German physician, proposed this apparatus in 1847.† However, it is to Brown-Séquard that we owe the demonstration that a platinum-tipped cautery is superior to the ordinary instrument, because it does not become oxidized and rough.

Cautery irons made of steel, with variously shaped tips, are still generally employed by surgeons and by veterinarians, but physicians, who see most of diseases of the nervous system, are unanimous in their approval of the platinum tip.

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* Reprinted from *Archives of Medicine*, April, 1879.

† Hoppe. Das Feuer als Heilmittel, oder die Theorie des Brenneus in Heilkunde; Bonn, 1847; cited in *Dict. des Sciences Méd.*, t. xiii., p. 408.

The methods of using the cautery have been different at various times, and in the hands of various physicians and surgeons. The ancient method, which prevailed up to about 1830, and is still in vogue, was to burn deeply; to use force in applying the instrument, and to thus produce a slough which separated with suppuration. This severe application was made purposely, in accordance with the prevailing doctrines, which taught that suppuration was useful for the removal of disease. In late years deep cauterization has been used under the erroneous impression that a greater degree of irritation was thus produced.

It is frequently stated, and I myself taught, that superficial cauterization, the method now chiefly employed in medicine, was introduced by Brown-Séquard. This is an error, since Jobert* (de Lamballe) as far back as 1838 described his "*cautérisation transcurrente.*" Valleix† used superficial burning in the treatment of neuralgia about the same time and later; and an elaborate essay upon the same mode of application (more especially for neuralgia) was published by Nota‡ in 1847, before Brown-Séquard had become engaged in the practice of the specialty in which he has since achieved such fame.

Jobert, Valleix and Nota held that the cautery (made of steel and intensely heated) should be applied very lightly, in parallel strokes, over the nerves which were the seat of pain. They deprecated destroying the skin and causing suppuration. Their results in neuralgia were very remarkable.

At the beginning of his practice, Brown-Séquard used the older severe application, and the moxa (treatment of Charles Sumner). Thus it appears that superficial cauterization as practiced by Jobert and Nota, had fallen into disuse prior to 1870.

I come now to my own knowledge of the cautery and its application. When I had the privilege of studying with Brown-Séquard, in Paris, during the winter of 1869–70, he taught me the use of this counter-irritant. He was employing it daily in various diseases, organic and functional, of the nervous system, with apparently admirable results. His instrument consisted of an olive-pointed steel cautery iron, about thirty centimetres long, the olive being about 15 mm. in diameter at the base, and carefully covered with platinum. He heated this instrument almost to a white heat in a grate fire, and applied it with extreme rapidity and wonderful lightness and certainty of touch to the scalp, mastoid regions, back of neck, spine, track of various nerves, etc. He taught me that superficial counter-irritation was preferable for several reasons: 1, The greatest effect upon nerves was thus obtained, because the terminal filaments and terminal organs of sensory nerves are more sensitive than their trunks; 2, Prolonged pain and suppuration were avoided; 3, Patients were able to go about immediately after the operation. He also insisted upon the use of platinum-tipped cauteries because their surfaces did not oxidize and scale as did those of steel, and thereby a smoother and more superficial burn could be obtained.

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* Jobert. Etudes sur le système nerveux. Vol. ii., p. 648, Paris, 1838.

† Valleix. Traité des Névralgies, Paris, 1841.

‡ Nota. *Union Médicale*, 1847. Tome i, p. 494.

In 1870 Messrs. Tiemann of this city made for me, after my indications, platinum-tipped cauteries, similar to Brown-Séquard's, and I used them until 1876 with satisfaction. Since 1872–3 they have been made by all instrument makers; some olive-pointed, others button shaped, etc. Fig. 1 represents the olive-pointed platinum-tipped cautery.

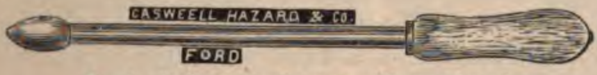

Fig. 1.—Platinum-tipped cautery of Brown-Séquard.

The only serious drawback to the use of this form of cautery is the mode of heating. In order to obtain almost a white heat, a strong bright grate or range coal-fire is needed. In winter this can be had at all times in private houses and in our offices, but in summer the physician is obliged to have a fire made purposely, or to take his patient into the kitchen (as I have often done). Besides, grate fires are not to be had in hospital wards. Another disadvantage of this cautery is that through repeated heating the iron part of the instrument is gradually worn away, so that the platinum cap ultimately becomes quite loose. In other respects this instrument is excellent; it is well balanced, and can, after some practice, be applied most lightly, so as to merely shrivel the cuticle; it is sufficiently small to be carried anywhere with other instruments, or in a deep pocket.

In 1876 a better instrument was introduced here by my friend, Dr. James J. Putnam, of Boston. This, the compound blowpipe gas cautery, a modification of an English instrument, is represented in Fig. 2.

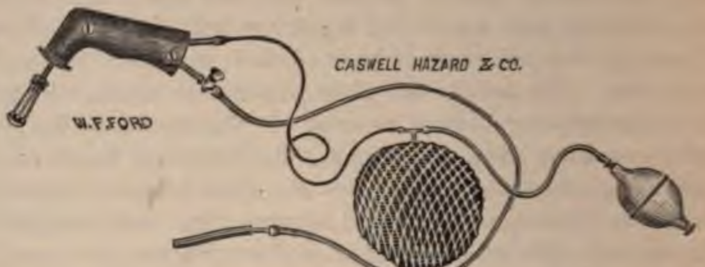

Fig. 2.—Gas blowpipe cautery of Dr. Putnam.

The instrument was exhibited before the American Neurological Association at its second annual meeting, June 7th, 1876, and a description of it was published with the Proceedings of the Association, in July, 1876.* I quote Dr. Putnam's concise description of the instrument: "It consists of a compound blowpipe with an appropriate handle, to the end of which a small platinum cup, fastened by three stout platinum wires to a brass collar, could be attached. A long and fine rubber tube ending in a brass tip for attachment to an ordinary fish-tail burner, carries the gas; a steady stream of air is pumped in by the aid of a rubber hand-ball and a receiver of very distensible rubber, in fact, an ordinary toy balloon covered by a good-sized bag of netted twine.

"The advantages of the instrument over others, consist in its adaptability to almost any place where it is desirable to use the cautery, and the ease with which almost a white heat can be maintained for any length of time."

I soon procured one of these instruments, and used it with great satisfaction for nearly two years. For practitioners in cities and large towns it is very available, as it can be set going wherever illuminating gas can be procured. For physicians in small towns and in the country it is useless.

Last year, however, a still more perfect and universally applicable instrument was placed at our disposal. Paquelin's benzine cautery (Fig. 3), designed for surgical purposes, is admirably adapted to medical uses. It consists of a straight handle containing a blowpipe, and bearing a variety of platinum tips. Those for surgical purposes are pointed or flat in coarse imitation of knives. The tips suited for counter-irritation are the olive and the button. The olive or cone in my own instrument measures 22 mm. in length, and 12 mm. in diameter at its base. I had one side of it turned up so as to make the olive somewhat keel-shaped in one profile.

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* The *Journal of Nervous and Mental Disease*, vol. iii., 1876, p. 434.

The button-shaped tip is like a miniature cook's cap, measuring 13 mm. across the top, and 8 mm. in height. The upper rim of the cap projects nearly 2 mm., and with it I am enabled to make extremely narrow linear cauterizations. Each tip is fastened to a metallic collar which screws on the handle. Through the handle a stream of mingled air and benzine vapor is forced by means of a hand-bag and balloon. The balloon serves to make the current of air steady and continuous. From the balloon the air passes into a bottle containing benzine. The whole instrument is packed by Messrs. Tiemann in a small box.

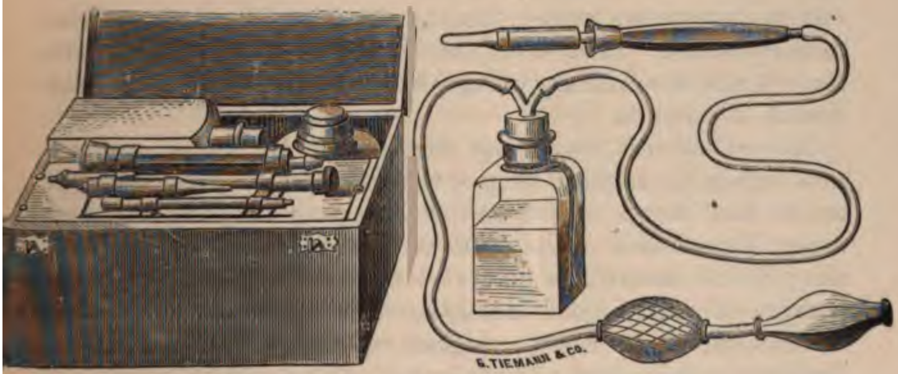
G. TIEMANN & CO.

Fig. 3.—Paquelin's benzine platinum cautery.

The Paquelin cautery is managed as follows: After screwing on one of the tips, its platinum end is held in a flame—gas, candle, alcohol, or coal fire—until hot, and then air is pumped through the instrument. The mingled air and benzine ignite, and are projected as a flaming stream against the platinum tip; and when this has become heated to a certain degree, a white heat is easily maintained after the removal of the instrument from the gas flame or fire. This increased combustion of benzine against heated platinum is very remarkable; the process can be kept up for a long time. It is important, I should add that the refined benzine sold in drug stores for cleansing clothing is too volatile to heat well; the commonest benzine is the best. In the last few months I have employed this instrument altogether in my private practice, though still employing the Brown-Séquard cautery and Putnam's gas cautery in my clinic. It is at once evident that the Paquelin cautery is superior to all others in its being available anywhere, in town or country, providing the operator be supplied with benzine, and a flame or strong fire to start the combustion. Its cost is great, but probably before long a simple form of cautery, with one tip, may be supplied for less than twenty-five dollars.

There is yet a fourth form of cauterizing instrument employed, viz., the galvano-cautery. I have not employed it, but recognize in it one advantage; it is noiseless. The gas and benzine blowpipe cauteries make a hissing sound, which greatly alarms many patients. Obvious objections to the galvano-cautery are its cost, and the difficulty of carrying the whole apparatus to the homes of patients.

In conclusion I would say that any of the above cauteries will suffice for medical purposes; they all satisfy the requirements laid down by Brown-Séquard; they are susceptible of being heated to a white heat, and being made of platinum, they remain smooth in spite of long usage. The choice of an instrument must be made chiefly upon the non-essential grounds of convenience and fancy. A glass rod heated in a spirit lamp has been proposed as a handy substitute for a cautery. It is a very inferior instrument, chiefly because it cannot be made very hot, because it bends and becomes unmanageable when hot, and because it cools very rapidly.

I now pass to a brief statement of the methods of applications.

Usually I follow the procedure called "*cautérisation transcurrente*" by Jobert and Nota, and adopted by Brown-Séquard, viz., I make very light parallel strokes with the cautery at white heat over the part chosen as the seat of counter-irritation. I aim to affect only the cuticle, and try to avoid subsequent suppuration. From four to twelve strokes can be made in an incredibly short space of time, and with very little suffering. The patient should be carefully placed with reference to the light and the comfort of the operator, and in many cases it is well to tell the patient to hold a handkerchief over his eyes. One error to be avoided is striking hard at the beginning of the strokes, since that is certain to cause blistering and suppuration. Of course the best skill is baffled by sudden movements of the patient. The strokes should not cross one another, since at the point of crossing too much effect would be obtained.

The only dressing required for a successful burning is a thin piece of old muslin or linen, which is to be pinned or sewed to the patient's underclothing. Burns behind the ear, or the upper cervical region, and on the face, require no cloth. In case of suppuration, simple cerate, carbolized cerate, or vaseline may be applied twice a day.

It is often desirable to repeat the cauterization frequently. In spinal affections, for example, we may begin at the top, and by means of daily or tri-weekly applications, cauterize the entire spinal region systematically and repeatedly.

With reference to the pain of the operation, patients differ greatly in their account of the sensations produced. The majority of my patients have considered the pain slight, and speak of its disappearance in twenty minutes or an hour. A few patients claim they suffer very much, and an equally small number declare that it is not at all painful, less painful than dry cupping. It seems quite clear to me that in most persons less pain is produced by the cautery than by a blister. Dr. Hammond recommends freezing the skin previous to applying the cautery, but this seems to me highly unphysiological. I cannot here enter into a consideration of the *modus agendi* of counter-irritation, but can simply state that the modern doctrine of superficial counter-irritation involves *irritating* the terminal nervous twigs and organs, and obtaining distant reflex vascular and nervous effects. Now, impairing or abolishing the irritability of the nerves of a region we are about to cauterize, seems hardly logical. It might be defensible for the older deep application, which was believed to relieve by the subsequent suppuration. I certainly advise cauterizing the active sensitive skin.

The immediate result of the burning is a brownish welt with some roughness, due to shrivelled epidermis. Very soon a zone of hyperæmia appears around the streak, and lasts for a long time; hours in some cases. When several parallel strokes have been made close together upon a part, an enormous cutaneous hyperæmia results. This increased circulation is probably a part of various vascular movements produced by the application, and necessary to its efficacy. Later results are a dry, brown scab, which falls off in a few days, leaving a red or reddish-brown scar, which disappears surely but slowly in almost every patient.

The popular dread of the cautery is great, chiefly because of certain absurd accounts of the burning of patients which have appeared in the secular journals, and partly because the terms used—"burning," "cautery,"—are alarming to all but most placid minds. Yet I do not think that it is right to cauterize patients by surprise, without a warning and explanation. I always tell my patients what I mean to do, how I mean to do it, and what the usual estimate of pain is. It is very seldom that I meet with a refusal to allow a first application, and after this trial no objections or complaints are heard as a rule. I have applied the various forms of cautery to persons of all ages from eleven to above sixty years, to both sexes, to persons of extremely nervous temperament, and in most various pathological conditions, and my experience has been so satisfactory that I now use the method more than ever, and consider it a more valuable mode of counter-irritation than any other.

This account of the actual cautery has become so extended that I cannot do more than enumerate the diseases in which I have employed it, or known it to be employed with success. Superficial cauterization by the actual platinum cautery has been satisfactorily used in the following conditions:

1. Neuralgias, acute and chronic, of the trigeminus, and of [illegible] peripheral nerves.

2. Spinal irritation, and the various cerebral paræsthesiæ (pressure, numbness, etc.,) whose pathology is now obscure.

3. Spinal congestion.

4. Various forms of myelitis, acute and sub-acute.

5. Epilepsy (not by myself).

6. Intercostal pain.

7. Lumbago, acute and chronic.

8. Articular inflammation. (White swelling of knee by Dr. McBride; traumatic arthritis of wrist, by myself.)

9. Peri-arthritis (chronic rheumatism?), especially of the shoulder.

In general terms the cautery is a potent and harmless substitute for blisters and pustulating ointments, in the various affections where counter-irritation is called for.

I trust that I will not be misunderstood as holding the actual cautery to be a panacea, and as urging its indiscriminate use. It is a remedy which, like all others, is to be employed only when indicated. The promiscuous burning of patients who present themselves with obscure nervous symptoms, or who are hypochondriacal, I unhesitatingly condemn.

TRAUMATIC PEDAL NEURALGIA OF ONE YEAR'S STANDING RAPIDLY CURED BY THE ACTUAL PLATINUM CAUTERY. *

Dr. K——g, aged 50, seen December 25, 1877. Is a man of good constitution; never subject to neuralgia. A little over two years ago one wheel of his wagon passed over the end of his right great toe, producing a moderate bruise but no fracture, luxation, or cut. In a few days he was perfectly well. In the last twelve months has suffered from gradually increasing pain in the toe which was injured, and along the inner side of the sole of the foot as far back as the ankle. The pain is burning, pressing, aching, not lancinating. It is worse in the daytime, and is aggravated by using the foot. No numbness or anæsthesia has been observed, but, on the contrary, there has been great hyperalgesia of the affected region, with some tumefaction and great hyperæmia. No pain above ankle; but patient has "fancied" that he had slight "sympathetic" pains in the left great toe and in the pulp of the right thumb, when the pedal neuralgia was greatest. No head symptoms; no signs of paraplegia; bladder normal. No gout. Has been confined to the house for four and a half months.

Examination.—Right great toe and inner half of foot tumefied and red; the veins are large and there is much capillary stasis. No nodosities or other lesion exist about the affected toe. No true neuralgic tender point can be discovered, but some time before Dr. K. discovered one beneath the internal malleolus, near the sole. The whole right toe is very tender, and the chief pain is experienced along the internal aspect of the toe to its point.

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* From *Archives of Medicine,* June, 1879, vol. i.

The left great toe is rather reddish but not tender. When the patient stands the passive congestion becomes enormous, and extends almost up to the groin. Repeated measurements by Dr. K. and myself show that the right foot (back of toes) is .5° C. hotter than the same part of the left.

The doctor bears the facial expression, and has all the attitudes of one who has suffered greatly from neuralgia. He has tried nearly all remedies and applications without relief.

Diagnosis.—Traumatic neuritis of branches (and trunk?) of the internal plantar nerve.

I employed the actual platinum cautery applied over the posterior tibial nerve behind the malleolus, and on the seat of pain; no medicine was given. The first application gave relief; after the third burning Dr. K. was able to walk, and after the sixth the neuralgia disappeared; these cauterizations were made at intervals of two days. The great hyperæmia of the lower extremity continued. Dr. K. resumed his practice.

Early in May, 1878, a slight relapse occurred, which was completely cured by two or three applications. Since that time there has been no return of neuralgia, though the toes are the seat of slight semi-painful or cramp-like sensations. The hyperæmia had almost disappeared by autumn. In the past year the patient has not lost one day from neuralgia.

CASE OF DESQUAMATION OF THE KIDNEYS DURING THE ADMINISTRATION OF MERCURY AND IODIDE OF POTASSIUM.*

A YOUTH of 19 was under my care during the last half year for symptoms of cerebellar tumor, viz., epileptiform attacks, occipital headache, partial opisthotonus, moderate neuro-retinitis, partial right hemiplegia, ending in death. The urine was examined in the autumn and found normal. In December, 1878, the occipital pain and spasm became very severe, partial right hemiplegia showed itself, and the patient was given the red iodide of mercury, .01 gram, and iodide of potassium, 2 grams, four times a day; blisters were applied behind the ears. Improvement showed itself early in February, and about that time the urine was examined because of its great quantity. What was my surprise at finding in it an enormous number of hyaline and epithelial casts. There was no albumen, and the patient had no other symptom of renal disease. From February 13th to March 7th, numerous examinations were made, with the following results: urine of good color, specific gravity varying from 1,018 to 1,020, absolutely free from albumen, depositing innumerable casts of all sizes, hyaline and epithelial. In some instances eight or ten casts were counted in a field. Prof. A. L. Loomis examined two slides containing these casts. At Dr. Loomis' suggestion the medicines were stopped February 17th. Until February 24th, casts were still detected in diminishing number, hyaline casts being the last observed. The iodine reaction also persisted for at least a week after the stoppage of the iodide. On March 7th, it is noted that no casts are to be found, and this negative result was obtained on several other days.

About March 24th, after a remarkable remission of two weeks (walking about the house), the epileptiform attacks, opisthotonus, and severe occipital pain recurred, and the mixed treatment was once more begun. The amount of mercury and iodide of potassium given in this relapse was about the same as before (.01

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* From *Archives of Medicine*, June, 1879.

gram and 2 grams four times a day), with extra doses of iodide alone afterward. On some days the patient took nearly 15 grams. On April 9th, the urine had a specific gravity of 1,018, contained 1 per cent. albumen, and an immense number of hyaline and epithelial casts; also free renal epithelium. As before there were no symptoms of renal disease. Several other examinations were made previous to death on April 18th, with the same result as regards casts. The last two days were characterized by a steadily rising temperature up to 41.26° C.

The symptoms in life, and the mode of death, indicated organic cerebral disease, whether a chronic basal meningitis or a cerebellar tumor we were unable to decide, as a post-mortem examination was refused.

The most interesting point in the case was the occurrence of severe renal desquamation on two occasions, apparently caused by iodide of potassium, or by it and mercury combined. That a few hyaline casts may appear in the urine of patients taking iodide is generally known, but the shedding of enormous quantities of epithelium was startling. It should be remembered that the first desquamation ceased about one week after the iodide was stopped, and that the iodine color and the casts disappeared about the same time.

My friend Dr. F. P. Kinnicutt communicates another case which occurred in his practice. I quote the doctor's notes: "The patient was taking .003 gram of biniodide of mercury and .75 gram of iodide of potassium three times a day for secondary syphilis. On March 23, 1877, he contracted an urethritis. Examination of the urine showed numerous leucocytes and hyaline casts. The latter were of varying size, but all of similar character. There was a mere trace of albumen, easily accounted for by the number of pus corpuscles. With the complete cessation of the urethritis (in three weeks), the trace of albumen disappeared, not to return. Hyaline casts, however, continued to be found in great numbers. On May 20th, the casts were still present in the same numbers. The iodide of potassium was discontinued, but the mercury was not omitted. Within a week after the discontinuance of the iodide, the casts were fewer in number, and early in July had notably diminished. Patient went into the country at this date; on his return in November a careful examination of the urine failed to show a single cast. [Mr. B. had been under my observation previous to his having contracted specific disease; his urine had been examined and found healthy. During the period that casts were found the patient's general condition was excellent in all respects. The urine has been examined from time to time up to this time, and at no time have any casts been found. The iodide of potassium was never resumed.]"

It would seem from these two cases (to which I might add a third now under observation), that iodide of potassium may give rise to the formation of hyaline and epithelial casts, without albuminuria or rational symptoms of Bright's disease.

"FOLIE À DEUX."

AN INSTANCE OF APPARENT CONTAGION OF INSANITY (MELANCHOLIA).*

K. L., aged 28, a single woman, seen in consultation with Dr. A. Jacobi, May 19th, 187–. There is a complex history of nervousness, hypochondriasis, and approach to hysteria in the last two years. Lately well-marked hypochondriacal melancholia, with constant talk of disease of the brain, extraordinary symptoms, etc. Patient has the facies and manner of a hypochondriac. Inquiry has revealed the important fact that for some time patient has practiced self-abuse, and has endured great self-reproach in consequence. Seen again on June 3d. Is in full acute melancholia, refusing food, attempting suicide, not sleeping, growing weaker. Sent to asylum.

P. L., a sister of the preceding patient, aged 25 years; was seen on June 12th. During the illness of K. L. she had become nervous and depressed, and was removed from home to some friends. But she there grew worse, and at the time of K.'s commitment (June 3d) she was decidedly melancholic; reproaching herself for having caused her sister's ruin, declaring that she was not fit to live, etc. It appears that these two young women had slept and masturbated together. When seen June 12th, fully developed acute melancholia is observed; is worse than her sister. Sent to the asylum.

The mother of the patients had once been insane.

It is interesting to add that both these patients recovered within eight months, and are now well.

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* From *Archives of Medicine*, June, 1879. Vol. i.

These two cases well illustrate the ætiology of *folie à deux*, or contagious insanity, as explained recently by Falret and Lasègue in an important memoir.* It is not the exhibition of the insanity of the first party which causes the insanity of the second party; in other words there is not a true contagion. Both parties must be predisposed to alienation, must live in the same moral atmosphere, be exposed to the same exciting causes, and experience similar or corresponding emotions and conceptions.

These conditions of apparent contagion, or nearly simultaneous development, were found in all of Lasègue's and Falret's cases, and they are well exemplified in ours. Both the patients were children of an insane parent, both had enjoyed certain emotions and committed physical excesses together, both felt acute remorse for the vicious indulgence, and the second suffered in addition moral torture from the notion that she was responsible for her sister's illness.

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* La folie à deux, ou folie communiquée. Annales Médico-Psychologiques, Nov., 1877, p. 321.

CASE OF HEMIPLEGIA WITH FIRST SYMPTOMS IN THE FOOT, AND A LIMITED CORTICAL LESION.*

I VENTURE to add this imperfect case to the admirable one reported by my friend Dr. Miles, because, with certain allowances, it may serve in the discussion about the effects of cortical lesions.

In November, 1878, I saw, in consultation with Dr. Granniss, of Saybrook, Ct., a gentleman aged 54 years, who was hemiplegic on the left side and almost unconscious. The following account of his illness was furnished:

In December, 1877, after having enjoyed good health, he awoke one night with clonic convulsions of the left toes, foot and leg only. There was no impairment of consciousness, no spasm in any other part. He watched the spasm some time, and made comments on it. Since, there has gradually developed a left-sided hemiplegia. For months only the foot and leg were paretic; in the last few weeks the left arm has become weak, and now the left cheek is paretic, though the relatives have not noticed it. In January, 1878, vision became impaired, but an examination by Dr. Noyes revealed no cause. In the last few weeks patient has seen double at times, and sight has gradually failed. Severe headache has existed from the first; frontal, bilateral pain, most marked on the right side. This pain has been worst about daylight. In the past month pain decidedly nocturnal. On a number of occasions "lost himself" while out of doors, not remembering where he had been (petit-mal?). A business associate thinks that patient has committed errors in judgment. No extravagance in design or in deed. Lately has become stupid and semi-comatose.

Since January, 1878, a tumor-like swelling has appeared over the right parietal region. No albuminuria, but has had several attacks of gout. After severe cross-examination, patient admits having had a chancre, fifteeen years ago, treated with mercury; denies secondary and tertiary symptoms.

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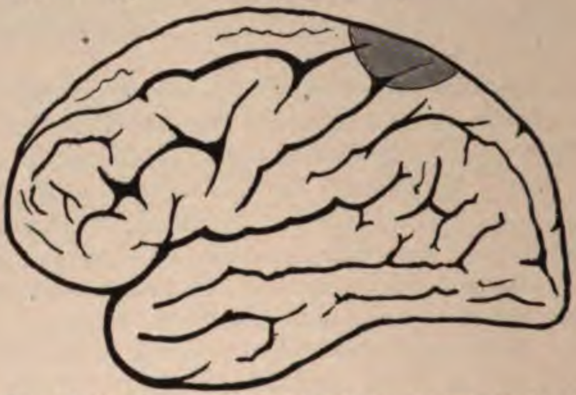
* From *Archives of Medicine*, Aug., 1879.

Examination showed a typical left hemiplegia, face and limbs. No diplopia, pupils small and equal; after atropia there is found a well-marked double neuro-retinitis. Sensibility preserved on the paralyzed side. Articulation indistinct; no aphasia. Stupor is peculiar, like that of drunken sleep. Patient can be roused by loud talking and shaking, and then answers correctly (showing fair memory) and clearly. The swelling upon the head, raised perhaps half an inch, is just above the right parietal eminence, extending inward to the median line, and forward almost to the vertical line from the meatus auditorius to the bregma. This tumor overlies Ferrier's centres for the legs.

Diagnosis.—External and internal nodes involving dura mater and the subjacent gyri of the right hemisphere.



A few days later the patient died comatose, and after much trouble Dr. Granniss secured a partial autopsy. He was not allowed to raise the brain from the skull or to incise it. He simply removed the calvarium and noted the lesions at the vertex. He found that there was an internal as well as an external osteitis, forming quite a tumor, which had, after adhering to the dura, exerted great pressure upon the subjacent convolutions. Dr. Granniss marked the location of the cortical lesion upon an Ecker's diagram, and the annexed wood-cut is a copy of his sketch.

It is of course very much to be regretted that a thorough examination of the brain was not permitted, but in view of numerous recent cases, it is impossible not to admit a causal relation between the lesion causing pressure upon the inner end of the right ascending frontal and parietal convolutions and the symptoms in the left foot and leg—spasm and paralysis.

CASE OF SLOW PULSE AND EPILEPTIFORM CONVULSIONS.*

H. G., aged 43 years, seen in consultation with Drs. Wm. Detmold and S. S. Jones in September, 1875. Present illness dates back more than one year. In former life, in adult age had frequent attacks of "glimmering before the eyes," lasting from a few minutes to half an hour, and invariably followed by headache, lasting nearly all day. Never observed absolute blindness in these attacks. These seizures have become fewer in the last few years; none in eight months. In the spring of 1874, patient consulted Dr. Detmold on account of a number of severe epileptiform attacks, without biting of tongue and subsequent drowsiness. It was then for the first time observed that the patient's pulse was very slow, from 24 to 28 beats per minute. Under tonics the pulse-rate increased slightly.

The patient describes some of his attacks as consisting chiefly in a want of breath, and severe constriction about the chest; no actual pain, and no symptom of angina pectoris. In some attacks he is a little dizzy, and even loses consciousness for a moment. These seizures have been very frequent, day and night. Even in the worst attacks the patient has never injured himself. Dr. Jones saw him in one epileptiform seizure; he then seemed like a man struggling against asphyxia, had clonic spasms, and a bluish face; the pupils were not examined; consciousness was not perfectly lost.

Examination.—Patient is a medium-sized, spare and pale-faced man. He sits naturally, and walks slowly but well around the room. Features anxious and drawn, suggestive of hypochondriasis, no headache. Nothing abnormal about the head. Some ten days ago Prof. J. H. Knapp examined the eyes with the ophthalmoscope and found them normal. No paresis, numbness anæsthesia, etc., in the limbs. The pulse beats 26 and 27 per minute in two counts (not successive); the artery seems tense, and by the finger one would describe the pulse as *pulsus tardus et durus*, no enlargement of veins, or evidence of embarrassment in the capillary circulation. The heart's impulse is very faintly perceptible to the finger in the fifth intercostal space within the nipple line; the dullness area is not increased. The cardiac sounds are almost normal; the impulse movement (systole) is apparently a rolling one, with a slight, inconstant systolic murmur, best heard over the cartilage of the fifth rib between the outer edge of the sternum and the nipple. It is probably an unusual contact sound of heart against the parietes. At the apex of the right lung there are slight dullness, increased vocal resonance, and prolonged expiration. Much nervousness (even to crying) has been observed, and patient has been disposed to magnify all his symptoms. Urine normal; no sign of tumor in cervical region near the vagi.

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* From *Archives of Medicine*, Aug., 1879.

The pulse-tracings appended were taken with Marey's sphygmograph. Fig. 1 was made under a low degree of pressure and perhaps gives the better picture of the pulse, with its high tension, and attempt at an extra beat in one place. Fig. 2, made under high pressure, shows a less normal dicrotism, and an imperfect ending of the systolic impulse; tension less. Both tracings indicate a want of perfect regularity in time and form in the various beats.*

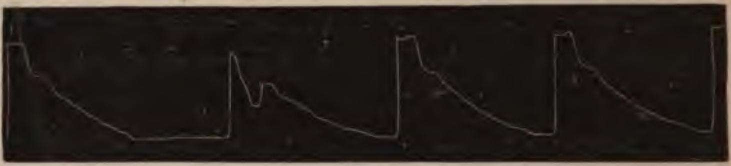

Fig. 1.

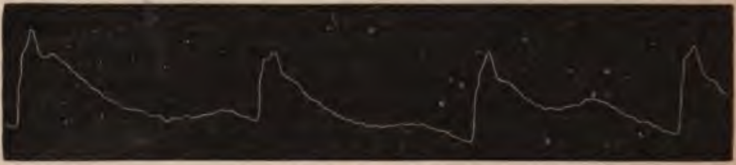

Fig. 2.

Remarks.—This case is remarkable, but not unique. Many cases are on record in which the pulse-beats ranged from 16 to 32 per minute. My friend, Prof. Cornil, of Paris, has placed on record † an instance of 14 beats; Landois, ‡ one of 10 beats. In several of these cases (Bradbury, § C. H. Jones ‖) "syncopal convulsions" and "epileptoid attacks" occurred.

The explanation of epileptoid attacks in cases of slow pulse does not appear difficult. The brain, and especially the basal masses, are rendered anæmic for too long a time. In other words, a degree of asphyxia (want of oxygen or excess of carbonic acid) is produced in these parts, and a discharge of nerve force takes place. The human being is thus placed in a condition resembling that of the animals whose subclavian and vertebral arteries were tied by Astley Cooper, Kussmal and Tenner. These observers first demonstrated by such experiments that anæmia of the basal parts of the encephalon will give rise to epileptoid attacks.

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* The lower diastolic part of the first beat in Fig. 1 is imperfect, because the lever descended too low and ran along the paper-holder for a moment.

† V. Cornil, Comptes-Rendus de la Société de Biologie, 1875. T. 2, 6^{me} series, p. 248.

‡ Landois, Die Lehre von Arterienpuls, 1872, p. 228.

§ Bradbury, Case of infrequent pulse and syncopal convulsions; death, autopsy. *Lancet*, i., 1877, p. 493.

‖ C. Handfield Jones, Lecture on slow pulse and epileptoid attacks. *Lancet*, 1876, vol. ii., p. 919.

It is singular that in H. G. we did not observe the Cheyne-Stokes' respiration rhythm, which is believed by many to be due to temporary asphyxia of the medulla oblongata.

We might add another case of slow pulse to this contribution, but, unfortunately, the notes and tracings have been mislaid. However, I can distinctly recollect that the patient, a male, about thirty-five years of age, was apparently not disabled by the slow action of his heart, though he had been somewhat weak for months prior to being seen. The pulse beat about 32 per minute, but the patient stated that it had been slower. No cardiac disease existed in this case, and no epileptiform attacks ever occurred.

In such cases it is a matter of surprise how the system at large can become accustomed to such a slow pumping of the blood. In both patients the principal organs, except the basal region of the brain in the first case, were normal; the lungs, liver, kidneys and various glands acting as well as if they received fresh oxygenated blood sixty or seventy times per minute. Calorification and the peripheral circulation were normal. In such cases we see a wonderful example of the self-regulating and accommodating qualities of a complex organism.

The causes of slow pulse are various. Some few individuals have a normal slow rate—down to fifty or forty beats. Again in some case of uræmic poisoning a very slow pulse may appear. Third, pressure upon the brain (clots, tumors, depressed bone) often reduce the pulse rate and raise the tension. But none of these pathological conditions can, so far as we know, bring the pulse down to the extreme figures referred to, viz., ten, fourteen, twenty-one, twenty-six, twenty-eight, or thirty per minute. Perhaps the best physiological explanation of very slow pulse is by irritation of the vagi in the neck, or more probably near their origin. In our two cases none of the above-mentioned conditions could be demonstrated.

A CONTRIBUTION TO THE PATHOLOGY OF ACUTE CENTRAL MYELITIS.*

THE case had a history briefly as follows: A male patient, æt. 33, unmarried, originally of good constitution, infected with syphilis, not conscious of straining his back or of catching cold, at 4.30 P.M., Oct. 8th was taken with pain in the lumbar region, located over the kidneys. Soon after he made ineffectual efforts to micturate; at 9.30 P.M. he made ineffectual efforts to empty his bladder, and was not able to sleep; at 11.30 P.M. he had great desire to micturate, and when he arose from his bed found that he was unable to stand. There was tingling in his toes and feet, and marked anæsthesia. The sensation at the knees was apparently perfect. He was restless, and his anxiety was great during the after part of the night. Pulse, 120; temperature, 37.8° C.

Oct. 9th, 3 P.M. Anæsthesia had extended upward to the level of the umbilicus; paralysis complete below. Catheter was used, an enema of soap and water was retained. At 8 P.M. he suffered from much pain in the sides of abdomen, lower part of thorax, and epigastrium. No symptoms referable to the hands or arms. Lower extremities absolutely paralyzed and anæsthetic. Spine only slightly tender in its medio-dorsal region; extremely restless; no dyspnœa.

Diagnosis.—Acute myelitis with softening, involving the entire thickness of the cord at a point in the lower dorsal region. The attack bore all the clinical characters of an acute inflammation from exposure to cold. He then suffered from a distressing sense of constriction about the body. On the fifth day of his sickness a strong faradic current was applied, one pole high in the rectum and the other over the abdomen, and an evacuation of the bowels obtained. In the third week there was notable atrophy of the muscles; and the level of the anæsthesia was at the ensiform cartilage. There was fever throughout the course of the disease, and the patient lived two months after the first development of symptoms.

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* By Dr. E. C. Seguin in collaboration with Dr. E. K. Henschel. Reprinted from the N. Y. *Medical Record*, Nov. 29, 1879.

A remarkable fact, relating to temperature, was that the temperature of the toes was higher than that of the body for the first few days.

Autopsy less than twenty-four hours after death: Enormous bed-sores over the sacral region. The spinal cord measured 32.5 cm., from the point of section, in the lower cervical region, to the *filum terminale*. The lower dorsal region was the seat of marked swelling and evident softening. The cord was carefully hardened, first in absolute alcohol, and then in bichromate of potassa; and, when examined microscopically, presented the following lesions: Descending degeneration of the postero-lateral columns; ascending degeneration; nearly complete destruction of the anatomical elements at the seat of the greatest softening. Several ganglion-cells contained vacuoles; vacuoles and contents apparently quite unaffected by reagents. The coats of the blood-vessels were thickened. There were granular bodies—whether they were the granulation corpuscles or not, was undecided. Neuroglia thickened. Acute central softening.

PARAPLEGIA IN SYPHILITIC SUBJECTS.*

I AVOID the common term "syphilitic paraplegia," for several reasons:

1. It is not very scientific, because the present tendency is toward an anatomical classification.
2. The relation between syphilis and existing paraplegia in a given patient is often a matter of great uncertainty.
3. Paraplegia, which improves under the use of mercury and iodide of potassium, is believed by many to be syphilitic, whether the patient admits or denies syphilis.
4. There are no definite symptom groups, which inform us that syphilis has attacked the spinal apparatus.

In the histories of four cases related the following were some of the interesting features which they presented: All four described chancres, and had secondary symptoms. The time which elapsed before the paraplegia began was variable, as follows: In one case *twenty-six* months; in one *six* months; in one, *ten* months; and in one, *seven* years elapsed after the development of the initial lesion before paralysis manifested itself. With one exception the development of the paraplegia was rapid. There was paralysis of the bladder in all four cases, thus indicating lesion in the dorsal region of the cord. The attacks were all severe. Complete cure was obtained in only one case. There was a remarkable amelioration of symptoms in all the other cases.

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* Read at meeting of N. Y. Neurological Society, March 3, 1879. Reprinted from the N. Y. *Medical Record,* April 5, 1879.

Two cases were then reported which illustrated mistakes in diagnosis. In one case there was the exceptional symptom, namely, *marked staggering when the eyes are closed, yet no anæsthesia of the soles of the feet.*

I think that at the present time we are not able to make a positive diagnosis of paraplegia dependent upon syphilis. Paraplegia of syphilitic origin is usually atypical. It was far from proven that no diseases were cured by mercury and iodide of potassium, except syphilitic affections.

The treatment of paraplegia occurring in syphilitic subjects should be energetic, and should be carried on by the simultaneous use of mercury and iodide of potassium. The iodide of potassium should be used after the American method—fearlessly; and even as much as 32 grams can be administered daily. Tonics were frequently required, and the best was cod-liver oil. It was important to keep the bladder empty so as to prevent or reduce cystitis, and to prevent bed-sores. In conclusion, I would call the attention of the society to the following points:

1. The question of diagnosis; was it possible? 2. The manner of giving mercury and iodide of potassium in this affection. 3. The value of the therapeutical argument in diagnosis, *post hoc;* and 4. The prognosis in paraplegia occurring in syphilitic subjects.

A CASE OF MOVABLE KIDNEYS; REMARKABLE VOLUNTARY CONTROL OVER THESE ORGANS.*

MRS. V., an American, aged about 31 years, consulted me on October 7, 1879, for "Nervousness" which had lasted eight or ten years. On examination I found she was hysterical, debilitated, dyspeptic, and that her uterus was moderately anteflexed and anteverted. Her last child was born four years ago. In the succeeding year, three years ago, she suffered for a whole winter from repeated attacks of severe hepatic colic, vomiting and subsequent jaundice; a few gall stones were seen in the fæces.

In the course of her detailed story, Mrs. V. mentioned that some time after these attacks of colic she had noticed "lumps" in her abdomen, and that they have been present ever since, making their appearance and moving about under her control. One physician had told her these lumps were "muscle," another that it was "the liver." They had never caused her any pain.

Examination of the abdomen in the recumbent posture showed a slim-built body, but little covered with fat; simple palpation showed nothing abnormal, deep pressure in the left side of the abdomen just below the ribs, revealed an obscure sensation of a rounded solid body. The patient now brought down her kidneys. By a powerful expiratory effort, drawing the lower ribs downward and inward, thus compressing the upper part of the abdominal contents, the organs made their appearance under the hand, and could be felt and grasped, they were globular, firm, not tender. The left kidney presented at a point distant 7 cm. from the median line, and about on a level with the umbilicus, or half way between the lower border of the ribs and the crest of the ilium. The right kidney escaped from under the lower border of the liver, and presented at a distance of 9 cm. from the median line; not descending much below the edge of the liver. Upon the cessation of the expiratory effort the organs disappeared from these locations. The left kidney is much more movable than the right.

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* From the *Archives of Medicine*, December 1, 1879.

The other organs of the abdomen seem quite normal in size and position. The urinary secretion has always been free—too free often.

The interesting points in the case are:

1. The occurrence of double dislocation of the kidneys.
2. The ability of the patient to make the loosened organs descend and present under the anterior abdominal walls.
3. The probable ætiology, through the strong muscular efforts attendant upon hepatic colic.

I may add that statements relative to the uterine and renal displacements were corroborated by my friend Dr. Paul F. Mundé.

CASE OF CEREBRAL HEMORRHAGIC PACHYMENINGITIS.*

Mr. X—, æt. 68, a merchant, seen on September 19, 1879, with Drs. Abbe and Herrick. The patient's general health had been excellent for years. In July of this year he had what was called a partial sun-stroke. About the last week of August returned from a trip to Colorado, looking badly.

A few days after return, a "neuralgic" headache set in on the right side, and has continued, with intervals of from two to five days, until now. Within ten days from the beginning of this headache a most remarkable failure of muscular strength, not of paralytic form, manifested itself. Pain has been very severe, and more generalized (less neuralgic) in the last three or four days; severe at night. Since the 17th (two days ago) great restlessness, physical and mental apathy and failing strength have been observed. No aphasia symptoms. A more important symptom has also appeared, viz.: a slight stiffness, with occasional clonic spasm of the right arm and hand. The right cheek has been thought flabby.

Mr. X. has been indisposed to use the right arm and leg, and has preferred to lie in bed. The pulse, in the last two days, has risen to between 70 and 80, after having been slower. The axillary temperature has risen to 37.4° C. Memory unimpaired no albuminuria. Pain in head now diffused and frontal.

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* Reprinted from the N. Y. *Medical Record*, Jan. 24, 1880.

Examination.—Patient lying on his back, with his right forearm and hand in the semiflexed and adducted position characteristic of contracture. Intelligence preserved; answers are slow, but correct, yet patient is indifferent and dull. Pupils normal; right orbicularis palpebrarum is the seat of constant clonic spasms, which extend to frontalis and other facial muscles. No twitching of right hand during the visit. Moves all limbs voluntarily, but extension of right arm and hand is incomplete, and there is marked rigidity (to passive motion) at elbow and in fingers. Mouth is drawn a little to the right (spasm); tongue is projected straight; articulation good; uses words correctly. No cardiac murmur, second sound hard, arteries tense; no redness on nates. In last two days involuntary, and, to a certain extent, unconscious micturition; patient is now wet without knowing it. No retention. Was always right-handed.

The symptom diagnosis was partial right hemiplegia, and it seemed to me that the most likely anatomical cause of such a peculiar paresis with hemispasm (face) was gradual interference with the blood supply of the motor area above the speech centre on the left side. I expressed the opinion that Mr. X. had atheromatous cerebral arteries, and that the terminal branches of the left Sylvian artery were blockaded. I anticipated gradually increasing hemiplegia.

I did not again see the patient alive, and I am indebted to Dr. Abbe for notes.

On September 20th, contracture of the left forearm and hand appeared, both arms being similarly stiff and semiflexed; they were occasionally drawn up by spasm. Vomited.

Sept. 22.—Increased drowsiness; both arms contractured, complains of head feeling hot inside (cool outside). Lucid intervals. No anæsthesia. Temperature in axilla 38.1° C.; pulse, 110.

During the last thirty-six hours of life the symptoms were profound drowsiness, absolute loss of vision. He occasionally whispered, and when asked if he heard what was said about him, said yes.

It is now stated that on September 18th (the day before I saw Mr. X.) he used the wrong words a few times, saying, for example, "diasnosis" for "diagnosis," etc. Mental action remained clear, but slow, until the last few days, when deep coma set in. During the last three or four days of life (even while some consciousness remained), sight was lost. To the very last of life the pupils were small and fixed. The pulse was abnormally full and slow from the onset of the disease (I found it *tense*), beating regularly from 58 to 60 a minute. At the time of consultation, six days before death, it was higher, nearly 80, and rose to 110 or more in the last three days. Slight fever was observed in the last few days.

The patient lived perhaps one month after the onset of the peculiar cephalalgia.

Sept. 25.—Died comatose.

Autopsy made 24 hours post mortem. Head only examined. While removing the calvarium two unusual phenomena occurred. When the sawing was nearly done a small hole was made in one temporal region through the dura mater, and from this, through the line of sawing a fine jet of dark blood came out with extraordinary force, not per saltum. When the skull cap was torn off (with great effort, as the dura was closely adherent to the calvarium), the dura gave way in the frontal region, and a perfect torrent of dark liquid was forced upward and backward, drenching the operator, striking his face, and some of it reaching three or four feet behind the head upon the floor. No one present (Drs. Herrick, Abbe, Amidon, and myself) had ever witnessed such an occurrence.

It was soon seen that this escape of liquid blood was from the cavity of an immense double hæmatoma; a large amount of fluid blood and some sheet-like clots lay in cavities formed externally by the dura mater and internally by a false membrane, united at its margins to the dura. There were thus two sacs extending over almost the whole of the superior and lateral aspects of the cerebrum. These sacs extended from the outer border of the anterior fossæ to the tentorium cerebelli, being thickest at about their middle, *i.e.*, over the fissures of Rolando and Sylvius, and the adjacent motor convolutions, thinning off in all directions. It seemed to me that there was but a thin sheet of clot and membrane over the posterior part of the third frontal convolution on either side. Judging from the violent irruption of blood which occurred on opening the skull, it seems evident that the liquid blood in the sac must have been under enormous pressure.

Another proof of this lay in the remarkable deformity which the cerebrum presented, the hemispheres in their upper median regions being concave instead of convex. The greatest depression below the normal level of the convolutions was estimated by those present at about 18 mm. The excitable districts on both sides—the centres for the forearm and leg, according to physiologists—were the seat of greatest depression. The speech centre seems to have escaped direct compression. The arteries at the base of the brain and in the fissures of Sylvius were normal. The arachnoid and pia were everywhere healthy; not opaque or adherent to the false membrane. The brain was not cut, as I desired to preserve the cerebral deformity which for an hour did not vary. In hardening, the whole brain was somewhat distorted and the depression made to seem less.

THE INTRA-BUCCAL METHOD OF FARADIZING THE LOWER FACIAL MUSCLES.*

In the last two or three years I have used a ready method of applying electricity to the lower facial muscles, which has not, I believe, been generally known or employed. The method is based upon several facts: (1) that the inferior facial muscles present well-defined motor points upon the buccal mucous membrane; (2) owing to the constant moisture of the mucous membrane, the application of a strong current is well borne; (3) that, in consequence, a better contraction is obtained by a given strength of current applied in this way, than when it is applied percutaneously.

It would be tiresome to enumerate the muscles which can thus be made to contract in a most complete way; suffice it to say, that all the muscles below the malar bones and the nose can thus be reached. A good reaction of the masseters may be obtained.

The instrument which I employ is figured in the annexed wood-cut. It consists of an ordinary interrupting handle, armed with a rod-like electrode of moderate length (10 cm. or more), bent at right angles near its extremity, and terminating in a ball 5 mm. in diameter. The whole of the rod or stem, except the ball, should be completely insulated.

The indifferent electrode (sponge) may be placed in one of the patient's hands, upon the back of his neck, or over the trunk of the facial nerve.

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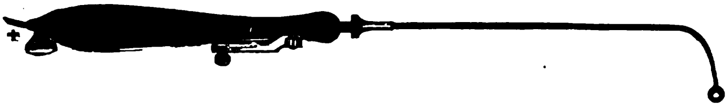
* Reprinted from the *Archives of Medicine*, vol. iv., No. 1, February, 1880.

The same special electrode will serve to make applications to the pharyngeal and palatal muscles.



By this method I have been able, in the stage of recovery of rheumatic facial paralysis, to obtain distinct contractions with faradism, when the strongest currents which could be tolerated on the skin of the face did not produce them.

ON THE COINCIDENCE OF OPTIC NEURITIS AND SUBACUTE TRANSVERSE MYELITIS.*

MR. PRESIDENT AND GENTLEMEN :—For nearly thirty years, since the first researches of Budge and Waller, of Claude Bernard and of Brown-Séquard, on the spinal innervation of the eyeball, physicians have been acquainted with various ocular symptoms of spinal diseases. The more prominent of these associations have been the myosis and atrophy of the optic nerves observed in the course of sclerosis of the posterior columns of the spinal cord, or progressive locomotor ataxia. Again, myelitis of the cervical spinal cord, whether inflammatory or from compression (Pott's disease, tumors, etc.), has been known to cause variable states of the pupil, due to irritation or destruction of the cilio-spinal centre, so-called, a region of anterior gray matter extending from the level of the fifth or sixth cervical nerve to that of the third or fourth dorsal nerve.

But the literature of spinal affections has been searched in vain for an example of transverse myelitis associated with an acute affection of the optic nerve. All of the recorded changes in the optic nerves in the course of spinal affection, were of a chronic and degenerative kind.

In the last year three instances of the remarkable coincidence of optic neuritis and transverse myelitis have occurred, and I thought it might prove interesting to lay them before you.

Although two or three cases had been observed and recognized by me before reading an account of the third, I think it but right to place this first in order of relation, because it was the first published. The observation is by the distinguished neurologist, Dr. W. Erb, now Professor in the University of Leipzig. His paper was read on May 17, 1879, at the fourth meeting of the Neurologists and Alienists of Southwestern Germany, held at Heidelberg; and it was published later in the autumn in *Westphal's Archiv.*†

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* Reprinted from the *Journal of Nervous and Mental Disease*, April, 1880. Read before the New York Neurological Society, March 2d, 1880.

† W. Erb.—"Ueber das Zusammenvorkommen von Neuritis Optica und Myelitis Subacuta."—*Arch. f. Psych. und Nervenkrankheiten.* Band x., Heft i., p. 146, 1880.

Case I., by Prof. Erb.—I was consulted, on July 18, 1877, by a man aged 52 years, who, previous to the present illness, had enjoyed good health, and had never had syphilis. He had experienced a combination of rapidly developed and peculiar blindness with alarming paralytic phenomena.

The following is a history of the case : In February, 1877, the left eye became suddenly affected ; diminished vision ; central scotoma, and in a few days total amaurosis ; and after a few weeks return of vision. The ophthalmoscopic examination was negative. Soon afterward the right eye was similarly affected. Blindness followed by recovery, negative results to ophthalmoscope. The beginning of the illness was marked by slight headache.*

After a while there was still another attack; this time in both eyes, commencing with bitemporal hemiopia and color-blindness; progressing rapidly to complete blindness. On this occasion the ophthalmoscope revealed a well-marked optic neuritis, with some distinct atrophy. At no time was there choked disk.

In the last few weeks improvement has once more shown itself. The patient can now read Jäger No. 4; distinguishes the outlines and colors of objects, but cannot yet recognize faces.

The treatment consisted in seventy-six inunctions with unguent. hydrargyri cinereum, local abstraction of blood, purgatives, and a seton in the neck.

During the three or four weeks preceding the consultation, there had occurred drawing and tearing pains in the legs, trunk, and in the lower thoracic regions (cincture pain); there was but little pain in the arms. In the course of fourteen days the following phenomena were added: Rapidly increasing weakness of the *right* leg, which soon became completely paralyzed, and at the same time anæsthesia of the *left* leg. Later still the left leg also became weak. Associated with these symptoms were retention of urine, later incontinence (now present) and anæsthesia of the urethra and rectum.

Examination on July 18.—The patient is a strong, healthy-looking man. He has slight fever (38.5° C.); amblyopia of both eyes; pupils and movements of the eyeballs are normal. The other special senses are normal. Memory and intelligence preserved; no headache or vertigo.

The upper extremities present no symptoms. The right hand is often the seat of slight pain, but there are no paræsthesiæ or disorders of motility.

The right lower extremity is entirely paralyzed, and the left thigh, though paretic, can be moved in all directions; the muscles of the abdomen and back are very weak.

The sensibility of the right leg is generally preserved, though in a few places it is diminished. The right half of the abdomen is evidently hyperæsthetic, as is a region round about the thorax at the level of the nipples. The left lower extremity and the left half of the abdomen are very distinctly anæsthetic; the left side of the back is anæsthetic, the right sensitive. These are the unmistakable signs of a lesion involving one lateral half of the spinal cord, as given by Brown-Séquard. The lower dorsal region is the seat of some pain; no spinal tenderness, or deformity, or stiffness.

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* The ophthalmic notes concerning the first stages of the disease, including the three distinct attacks of blindness, are by Dr. Steffan, of Frankfort-on-Main. These notes differ from Dr. Erb's summary in the important particular that in the first two attacks (each optic nerve alternately) a slight optic neuritis, œdema of edges, without swelling, was seen with the ophthalmoscope. Besides, some interesting limitations of the field of vision were noted.

Dr. Steffan will publish a full account of the eye symptoms in this interesting case.

The cutaneous and tendon reflexes in the legs are increased; reflex movement of abdominal muscles not present.

No atrophy or bed-sore. There is paralysis of the bladder; there is occasionally involuntary evacuation of urine; the patient is constipated, and he is not fully conscious of the passage of fæces.

Prescription.—Cold compresses, according to Priessnitz's method, to the spine; every three days dry cups along the vertebral column; iodide of potassium; extreme cleanliness, and attention to bowels.

July 27.—In the last few days signs of acute cystitis; bowels distended with gas; from time to time the legs jerk. Other symptoms not much changed. The right lower extremity is still completely paralyzed, the left a little weak. Sensibility very slightly diminished on the right side; there is no longer any hyperæsthesia; the cincture feeling is gone; the right half of the abdomen shows muscular tension. Plantar reflex and the tendon reflexes are greatly increased; dorsal clonus is easily produced. No bed-sore; eyes as before.

In the next few days, probably in consequence of the cystitis, there were several chills, and the temperature rose to 40.3° C.

In the next few days improvement began, and the following is noted on August 11: The left leg once more possesses all its movements and is quite strong; the right lower extremity is also movable, but is weaker than the left. There is hardly a trace of the alteration of sensibility; there are next to no pains in the legs. Reflexes less marked; bladder and rectum unchanged; eyes in *statu quo*. The patient's general condition is much better. Ordered same treatment, except that the iodide of potassium is omitted, and a little morphine given for insomnia.

Progressive improvement took place, so that on September 28th it is noted that the legs are strong enough to enable patient to take a few steps (no ataxia); the sensibility is normal, and the bladder acts well. At times he has a sense of tension in the back, and an occasional pain in the legs. The eyes have improved a little.

Toward the close of the year the patient's objective symptoms were about gone; the reflexes were still strong, but he complains of various paræsthesiæ in the legs, a "ringing" or vibration while sitting, sensations of weight and of swelling.

In the spring of 1878 the patient was well, except that he had sensations of slight heat and crawling in the legs and back.

From November 12, 1878, to March, 1879, the patient had a galvanic treatment for his eyes, with marked improvement.

Dr. O. Becker of Heidelberg, found the following: Slight myopia of both eyes; pupils rather small, acting well. R. V. $\frac{6}{60}$, L. V. $\frac{6}{36}$; fingers counted at 6 metres, with + 3 R. V. Jäger No. 3. L. V. No. 6. Both eyes are blind for green and red.

The ophthalmoscope shows atrophy of the optic nerves, with slight excavation; nerves bluish. Lesion more marked on left visual field, slightly reduced concentrically; no scotoma.

At the close of treatment, March 4, 1879. R. V. $-1=\frac{6}{18}{}_{12}$, L. V. $-1=\frac{6}{24}{}_{18}$, with + 3, can read No. 3 Jäger and make out a few words of No. 2; with + 4 and + 5 can read newspaper print easily.

CASE II.—Drs. H. D. Noyes and T. A. McBride. On September 5, 1879, I saw Mr. D., a patient of Dr. H. D. Noyes, at the request of Dr. T. A. Mc Bride. Dr. McBride has already made an exhaustive examination of the case, and the following is a history based upon a memorandum which he sent with the patient. I desire to express my thanks to Dr. Noyes and Dr. McBride for permission to make use of the case. Mr. D., a clerk, aged twenty-five years, suffered from debility during the whole of the past summer. Since March has had several "bilious attacks."

On August 9, was seized with severe diffused headache, which lasted day and night for a week. Was constipated and nauseated. No headache since.

August 18, retention of urine occurred, for which the catheter was used three or four times in the course of ten days. The bladder has been sluggish since. During the same period (last two weeks of August) patient noticed stiffness and pain in the muscles of the back, preventing his bending forward. The pain was in the lower dorsal region. In the last ten days no pain, but a sense of numbness and anæsthesia has appeared in all parts below the waist. The loss of sensibility was discovered in the bath; he did not feel the contact of water normally. About the same time (ten days ago) he also noticed a dimness of vision, which has since increased almost to blindness, at times. No symptoms in upper extremities. Patient denies syphilis, or injury to the head and spine. Several members of his family have died of phthisis. Examination: walks well; no disturbance of equilibrium, or inco-ordination. Dynamometer shows in right hand, 65, 63, 65; in left, 60, 56, 61 (weak instrument). No actual paresis of the lower extremities. Knee tendon reflex normal. Sole reflex deficient, especially on the right side. Sensibility is much impaired below the waist. Touch is badly perceived (an æsthesiometer point seems like a finger), and pricking or pinching still less. There is, consequently, more analgesia than anæsthesia. At times the legs tremble; no spasm, or formication. Sight is very defective, the fields of vision are irregularly limited, there is marked loss of color perception. The ophthalmoscope shows typical choked disk on both sides. The temperature in the mouth is 37.9° C. The heart is normal; percussion of skull and vertebræ produces no pain.

The above was Dr. McBride's examination. My own gave corroborative results, viz.: a paraplegiform anæsthesia (incomplete), and double neuroretinitis. The latter lesion seemed less than as described by Dr. McBride, and vision less impaired; he could count fingers and trace features easily.

My diagnosis was double lesion, one at the base of the brain, involving the optic nerves and the chiasm, and a focus of myelitis in the centre of the cord in its low dorsal region. I advised a continuance of the iodide of potassium in full doses.

[The following are additional notes furnished by Dr. Noyes. Dr. Noyes took part in the discussion on this paper, and exhibited to the Society diagrams illustrating the extraordinary changes in the fields of vision in his patient.]

"Vision became impaired at the same time that the bladder trouble came on. No phosphenes; no tenderness over lower portion of spinal column.

"September 2d.—The field of vision, O. S., normal. O. D.: Perception absent on nasal side, encroaching centrally beyond the median line, with contraction of the peripheral portion in other localities. Ophthalmoscope shows, O. D., the inner half of disk is most swollen—there is a small segment downwards and outwards, which is not much affected. It looks more like a neuritis descendens than a true choked disk. Not much choking of left disk. O. D. $H = 2.2$.

"The patient was next seen September 6th.—The condition of fields of vision being much the same, except that the sight has improved. Sight returning in the infero-nasal quadrant. O. S. normal.

"September 11.—To-day, for the first time, find that the left eye on the outer has lost its perceptive power almost entirely, there being only a small ovoid spot on the horizontal meridian where perception remains. The right now shows that the field is changed from the showing on the 6th, and things are reversed—seeing now only in the nasal quadrant. The patient feels satisfied that September 8th the change began for the worse in his left eye—at the same time that his right eye had changed as to field, viz., seeing only in the infero-nasal quadrant. The inner half of the right optic disk is swollen—the vessels are tortuous—the outer half is pale. The inner half of the left disk is swollen, the same as the right, the outer half being pale.

"September 16th.—His sight, in his own opinion, has not altered much. Examination shows recovery of a considerable amount of his lost fields of vision—the right being normal except for the presence of a scotoma; while the left shows the previously mentioned oval area of perception lower in the temporal portion of the field to have increased considerably in size; otherwise the field in O. S. is similar to last entry.

"September 20th.—Fields of vision have improved, there being only a central scotoma, of small size, in each field of vision. The sight is better, but it is not possible to measure it accurately. Both disks are in parts swollen, but not so much as at last examination—yet plainly to be seen still. There is an unusual pallor of other parts of the disks, that were at the earlier stages swollen.

"September 26th.—Patient says that his sight was much better yesterday than it has been for some time. His fields have not altered—the scotomata being still present. The color perception is poor. He recognizes blue and most of its shades. Red is recognized next. Gray, violet and green are mistaken.

"October 4th.—$V = \frac{6}{70}$ O. S.

"October 14th.—O. D. $V. = \frac{6}{200}$. O. S. $V. = \frac{6}{60}$. Cannot find positively any true scotoma in either eye. There is a certain amount of dullness of perception over the small scotomata found at the last examination.

"October 21st.—O. D. V. = $\frac{6}{30}$. O. S. V. = $\frac{6}{30}$.

"October 30th.—O. D. V. = $\frac{6}{30}$. O. S. V. = $\frac{6}{30}$. Inner half of both disks swollen—the outer pale.

"December 20th.—V. = $\frac{6}{20}$. O. D. No scotoma—color perception good. Fields of vision perfect for both objective and color tests.

"January 24th, 1880.—V. = $\frac{6}{20}$ in each eye. Fields for objective and color tests normal."

The paraplegia had long since disappeared.

Case III.—Personal.—Shortly before reading Prof. Erb's paper, I had the opportunity of seeing the following interesting case, and of treating it. The patient was originally under the care of Prof. Willard Parker, who, on December 9, 1879, transferred the case to me.

J. P. M., a banker, aged 35 years, had enjoyed excellent health for many years, and had never contracted syphilis. For some time previous to the development of the present illness he was in business in Virginia City, Nevada, at an altitude of more than 7,000 feet.

On September 5, 1879, he first noticed numbness in his feet and legs, but was perfectly able to walk. This numbness was stationary for three or four days; then a feeling was noticed as if there were an iron bar or block in the perineum; the legs became noticeably weak about the 28th. Mr. M. came east by way of Panama, and while on board the ship he used his legs actively. Arrived in New York in the first week of October; he could still walk to his meals in the hotel, though he dragged his feet—the right more. The numbness continued. He suffered a "distress" in the sacrum, but had no pain in back or legs. After a week, during which he exerted himself a good deal, the paralysis increased, and he ceased walking; sensibility became impaired. For a fortnight (middle of October) there was absolute loss of motility below the waist and much anæsthesia, though he never lost his feet in bed. At one time he had the feeling of numbness as high as the groins.

Sensation and motion returned in the left leg first; and since the end of October both legs have gradually but steadily improved. He can now move every joint in the lower extremities, but he has not yet tried to stand or walk. He has had a band-like feeling around the calves of his legs, and a pressure feeling in front of the abdomen. He never had retention of urine, but at times involuntary squirts. Was greatly constipated. There have been no active symptoms in the arms, but it was noticeable that if placed in an awkward position they easily became numb. The paralyzed muscles did not waste, no bed-sores formed, and the general health remained good. During the period of convalescence Mr. M. noticed severe tonic amid clonic spasms in the legs; less lately.

During the past two weeks blurred vision of the right eye has been noticed. This was preceded one week by severe pain in the right orbit and near the brow. Lately sensibility has greatly improved; a little tight feeling remains around the insteps.

Examination.—Patient is surprised to find that he can stand. Closing eyes does not impair equilibrium. The legs are weak, but every muscle and articulation can be moved. The reflex at knee and sole is exaggerated. Sensibility is normal to touch and pinching; localizes impressions correctly. No ataxia; the muscles are well nourished; spine not tender; erections (absent for a time) are returning.

Treatment was begun only at the time when paralysis became marked, seven or eight weeks ago. He was then given moderate doses of iodide of potassium, .002 of strychnia three times a day, and he was rubbed.

December 15th.—At my request Dr. Arthur Mathewson, of Brooklyn, saw the patient and examined his eyes. The following are Dr. Mathewson's notes: "On first examination the nerve of the right eye was found whitish and œdematous, with outlines rather indistinct; vessels only slightly tortuous, veins full and dark (in both eyes); media clear; refraction nearly emmetropic, but the most prominent part of the nerve disk was in focus with a + 2.2. Vision was not tested accurately for want of means at patient's house, but he could read about Jäger No. 10 with the affected eye. There was also a slight lateral tremulous motion of the right eye, a sort of nystagmus."

These two examinations justified the diagnosis of sub-acute transverse myelitis in the lower dorsal region, with optic neuritis limited to one eye.

I will not weary the Society with a transcript of my full notes of the further progress of the case. Suffice it to say that improvement in vision and in the power of walking, with decrease of reflexes, occurred, until at the present time the patient is nearly well. The treatment consisted in the withdrawal of the strychnia; the gradual increase of the iodide of potassium up to more than four grams three times a day, galvanism to the spine and muscles, and massage.

Dr. L. C. Gray, of Brooklyn, had the immediate management of the case, and I saw the patient nearly once a week. In January there was added to the above treatment an evening dose of two grams each of fluid extract of ergot and bromide of potassium, which had the desired effect of lessening the reflexes. On February 20th Mr. M. came to New York to see me. His gait was quite normal; the knee tendon reflex rather strong (no spontaneous reflex movements); he complained of only a trace of numbish sensation in the calves and in the nates; in walking a slight sense of constriction is experienced upon each leg below the knee, on the inner side. Vision of right eye is nearly normal; the nerve is whitish, [illegible] the nystagmus (horizontal) is still present.

March 1.—Dr. Mathewson has kindly sent me the following memorandum: "I have just carefully examined Mr. M.'s eyes as they stand to-day, and send you the result. There is now no limitation of the fields of vision, and no scotomata, and there is no marked diminution of color perception. The œdema of the nerve of the right eye has now wholly passed away, so that its outlines are perfectly distinct, and the disk is paler than normal, and quite in contrast with the nerve of the other eye, which is rather hyperæmic, with outlines not quite well defined. There is a manifest hypermetropia, of 1.5 (by ophthalmoscope 1.5 +) of the right eye, its vision is $\frac{6}{20}$, while the left is nearly emmetropic and has perfect vision also. There is still a slight trace of the nystagmic movement, though it is not constant."

The optic neuritis in this case was intermediate in type between the conditions observed in the two other cases. There was œdema of the periphery of the nerve with some swelling of the disk—a degree of choked disk. This was followed by atrophy without marked loss of vision. All the morbid processes occurred in one eye.

It is interesting to note that the distribution of the inflammatory lesions varied in each case within very considerable limits. In the eyes it affected alternately each optic nerve, and both at one time in two cases. In Dr. Noyes' case the changes in the fields of vision were singularly capricious. In the third case only one optic nerve was affected. These irregularities and the peculiar symptoms of bitemporal hemiopia (in Case I.) are, it seems to me, explicable only upon the supposition of a lesion at the base of the brain involving the chiasm and optic nerves. The phenomena in the third case (symptoms in one eye only) would seem to exclude most positively a central cerebral lesion.

In the spinal cord the inflammatory changes were in the dorsal region in all the cases, but in all other respects there were marked differences.

In Case I. the right half of the spinal cord no doubt contained most of the lesions.

In Case II. the æsthesodic region of the cord (posterior gray matter or peri-ependymal region?) was chiefly involved.

In Case III. the entire structure of the cord must have been slightly affected, the motor region most. The comparative escape of the bladder in Case III. (no retention) is instructive anatomically, as the limitation of the numbness to the altitude of the groin would indicate that the lesion was in the lowest dorsal or upper lumbar region of the cord, below the vesical centre. In Cases I. and III., where the limits of numbness and the constriction band indicated disease of the mid-dorsal portion of the cord, retention and cystitis occurred.

The question naturally arises: Is there any causal or physiological relation between the two sets of phenomena observed in these three cases?

Prof. Erb answers in the negative, and it seems to me that with our present knowledge of the relations between the optic apparatus and the spinal cord we must, in agreement with him, consider this association of optic neuritis and transverse myelitis as accidental.

A CASE OF MYSOPHOBIA.*

Miss X., aged 18 years, consulted me on January 30th, 1880, for a peculiar form of nervousness.

From the patient's mother I obtained the following history of the case. Childhood and girlhood had been healthy; menstruation began in the twelfth year, and has since been normal and regular. Patient has led an idle, luxurious life, doing as she pleased and taking a few private lessons at home. Has been allowed to rise at 8 or 9 A.M., to lounge about and read trashy novels. Was of a bright, happy temperament. The occurrence of insanity in the family is denied, though maternal grandmother had senile dementia, and one brother had fits at six months, followed by hemiplegia, epilepsy and imbecility. As bearing on the case it must be mentioned that patient's grand-uncle died of "cancer of the nose."

About three years ago patient had a moderate leucorrhœa, relieved by a tonic course. After this, was observed to droop and look tired, and she began to entertain the hypochondriacal fear or delusion that she too had or was to have cancer of the nose. (It was not till last summer that she confessed this notion, which was the cause of her inexplicable melancholia.) Ever since, Miss X. has been the victim of extreme hypochondriasis and mysophobia. The hypochondriasis consisted exclusively in fear of internal nasal cancer, and many of her first peculiarities,

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*Reprinted from the *Archives of Medicine*, vol. iv., No. 1, August, 1880.

as to washing herself and fear of contamination, were logically related to this idea; she fussed with her hair, face and hands, in order to prevent others from catching the cancer which she had.

It is useless to relate all the details of the morbid cleanliness of the patient. She would wash her hands every five minutes, and wipe each finger carefully and long. She would spend an hour or more in the bath-room, and use a dozen or more towels for her morning toilet. She would spend an hour or more in combing and brushing her hair. Great slowness in all acts is observed. Avoids touching door-knobs, plates, chairs, etc., without protecting her hand by a glove or a fold of her dress; refuses to pass dishes, etc., to others at table; will re-arrange a chair several times; has been seen to approach a door and retreat several times before passing through it; has stood in the middle of the room with an absurd automatic pendulum movement of whole body or of one arm; is reluctant to wear clothing, especially under-clothing, more than a day at a time.

Has bad, restless nights; at times is rather excited, and will pace up and down a room; usually inert, lies on a lounge with a novel; has seemed as intelligent as ever; at times pain in head and in mid-dorsal region; no hysteria or agoraphobia, or delusions of any other sort than the above.

The examination is nearly negative. Miss X. presents the appearance of health, and is rational. She admits the absurdity of her freaks and notions, blushes and attempts to hush her mother in the relation of details. Even as regards the original hypochondriacal notion of cancer in the nose she is not firm, having nearly lost her belief in that disease since a thorough examination of the nose and throat was made by a specialist some months ago. Explains her actions by her desire that no one should acquire the cancer from her. Hands are chapped by constant wetting; spine not tender; tongue clean; heart normal and pulse good. Has a semi-melancholic rather dull look, and eyes easily fill with tears. Denies masturbation.

I carefully laid out a plan of moral treatment, by which she was to be gradually and firmly prevented from doing her strange acts. She was ordered long walks and a little study. Every one near her was instructed to assume a semi-imperative tone and manner toward her. She was to clean her own room.

I decided to try a course of treatment by narcotics, similar to that which succeeds so well in mild melancholia. She was given extracts of opium and cannabis indica with a little rhubarb. February 24th great improvement is reported; patient has gained self-control and is cheerful; sleeps well. On account of nausea, the opium was omitted, and a pill of cannabis indica, reduced iron and rhubarb ordered. At bed-time 1.5 gm. of bromide of potassium. March 15th: Has been in the country two weeks, taking above remedies and walking a great deal. Is well. Advice: discontinue medicine, but occupations to be increased, exercise to be continued, and a firm moral hold to be kept on the patient.

I should add that Miss X. was not allowed to know the nature of her medicines.

While recording the case of this singular form of hypochondriasis, I desire to express my belief that a relapse, or the development of a different or more serious psychosis in the patient is very probable.

Mysophobia was first described and discussed by Dr. William A. Hammond, in Neurological Contributions, Vol. I., No. 1, p. 40. N. Y., 1879.

ON OCCIPITAL HEADACHE AS A SYMPTOM OF URÆMIA.*

I HAVE recently met with two cases in which occipital headache was so localized and persistent as to give rise to a strong suspicion of organic disease of the cerebellum, and in one of them a positive conclusion was only reached by means of a *post-mortem* examination. These cases both now appear to have been cases of contracted kidneys and uræmia.

I shall first relate the cases as they are in my case-book.

CASE I.—Lieut. X., U. S. A., aged thirty-six years, consulted me on November 5, 1879, and gave the following history: Until the time of his graduation from West Point he had suffered from frequent general headaches; but that since leaving the school in 1867 he had several severe attacks of occipital headache. These at first occurred two or three times a year, but in the last few years much more often, the attacks lasting from twenty-four to forty-eight hours, accompanied by vomiting and sometimes by delirium. These paroxysms were often relieved by bromide of potassium. In 1876, during Centennial times, he had one of his headaches, and with it an epileptiform convulsion, in which he did not bite his tongue. In February, 1879, at the same time with a headache, he had another convulsion, in which the tongue was bitten. He has noticed that while in the Northern States he has but few headaches, whereas when in Texas he has had a great many. He often had a feeling of soreness and fullness in the back of the neck, and is very nervous after the attacks; has been in the North since March. In August had a severe attack and another on October 26, aborted by bromide of potassium. This last headache was accompanied by stiffness and fullness in the back part of the neck. There are no special ocular symptoms during the attacks, and he considers his eyesight normal. During the paroxysms the face is flushed, the head feels full and pulsating. The father and the grandfather of the patient had sick-headache.

Mr. X. has abstained from the use of intoxicating drinks since 1876; he has never been injured about the head, and has never had syphilis.

Examination.—Eyesight normal to all ordinary tests; no astigmatism; no lesion seen with the ophthalmoscope. Cervical spine not tender; no symptoms of dyspepsia; heart normal. The general appearance is that of health. November 6th, looks puffy under the eyes. Three specimens of urine are examined with the following results: Their specific gravity is low, ranging from 1,018 to 1,020; they all contain albumen—from 1 per cent. to .5 per cent., and hyaline casts. The retinæ are re-examined with negative results. Subsequently, numerous examinations of the urine were made by Dr. Alexander, Surgeon U. S. A. at West Point, and evidences of chronic Bright's disease were invariably found, such as low specific gravity, hyaline and granular casts; the amount of urea in one period of twenty-four hours was about twelve grammes.

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* Reprinted from the *Archives of Medicine*, Vol. iv., No. 1, August, 1880.

CASE II.—Mr. J. W., a merchant, aged 47 years, was seen by me at Passaic, N. J., in consultation with Dr. J. C. Herrick, on December 21, 1879. I obtained the following history of the case:

The patient had formerly enjoyed good health; had never received any injury to the head; no syphilis. During all his adult life he has suffered from headaches, more or less periodical, perhaps one in three weeks, each attack accompanied by nausea, and usually lasting one day. Of late years he has had much less of this headache; it was evidently migraine. About twelve years ago, in the streets of New York, during hot weather, he had an attack which was called "sunstroke." The symptoms of this attack are unknown. Mr. W. consulted me in 1874, but I have no notes of his case except a memorandum of my examination of the urine. This appears to have been perfectly normal.

In the last two years he has been almost constantly suffering from some headache, a little every morning, and more and more often of late he has had severe attacks. In the past two months very severe headaches, with nausea and vomiting several times a week. During the last two weeks has been confined to his bed. The patient and his wife clearly distinguish this pain from the former headaches by several characteristics; the pain is more violent, it is distinctly occipital, and lately has been cervical as well; it appears in paroxysms at any time, chiefly during the day, and the pain itself is of a different character. After a migraine Mr. W. felt very well; but now after a severe headache he is prostrated and dull. The nausea always comes on after the pain; he has no nausea between the paroxysms. He has not had much frontal headache, but the pain has extended from the occiput into the vertex and the whole top of the head. Movements aggravate the pain. There is no affection of sight or hearing; no dizziness. Of late has needed morphia; Paullinia seemed effectual for a few days only. During the last week he has taken about 4 gm. of bromide of potassium a day, and on the day before yesterday he had 15 gm. in twenty-four hours. The attending physician has examined one specimen of urine, but found no albumen.

Examination.—Patient feeble; lies relaxed in bed; voice faint, but articulation is distinct; mind clear; the head is not tender. The right eyelid is in partial ptosis; no strabismus; the ophthalmoscope shows no lesion of the fundus (atropine used). The right side of the face is rather inert, but the tongue (heavily coated) points straight. The hands are of due proportionate strength; in walking the right foot is dragged after a few turns in the room. No incontinence of urine; morphia affects patient very readily; he has had none in twenty-three hours, yet he is dull, and his pupils are small and fixed. The heart is normal, but the pulse is quite irregular, beating 23 and 29 in successive thirds of a minute; twice in the minute an acceleration is noticed

There is a trace of œdema on the tibias. Patient denies that his neck is really stiff, though he carries his head on one side, and keeps it quite still; no opisthotonus. To-day the pain extends to the sixth cervical vertebra.

I declined to give a positive diagnosis until after the urine had been thoroughly examined. At the same time I saw that the patient was in great danger from exhaustion and a tendency to stupor; and that many of the symptoms of tumor in the cerebellum were present. One was lacking, viz.: neuroretinitis. I also thought he was brominized.

On December 23, three specimens of urine were received, and were at once examined by Dr. R. W. Amidon. The specific gravity was found to vary between 1,024 and 1,025; there was albumen in all, varying in amount from 3 to 10 per cent.; there were also in all specimens numerous hyaline and granular casts.

Mr. W. died on December 27th, in a comatose state; no convulsions or further paralytic symptoms having shown themselves. The autopsy, made on the 28th, showed that the cerebellum and the other parts of the encephalic mass were normal; while both kidneys were extensively diseased. The left kidney was found completely diseased, granular and hard in places; its membranes peeling off with difficulty. It had a reddened congested appearance, and showed some evidences of not only a chronic trouble, but of a more recent acute inflammatory action. The right kidney was found to be only partially affected; somewhat congested, and with the same type of lesion.

Dr. Herrick, to whose courtesy I am indebted for the above account of the autopsy, adds: "The results of our examination go to show evidently that, after all, the patient's symptoms may have originated from a renal disease, although we cannot explain yet why his headaches so many years should have been from such a cause. He had never complained of back-ache or of any of the usual symptoms of Bright's disease, except the head pain."

The following is a summary of the symptomatology of the two cases:

Both patients were adults; both had suffered from chronic headache more or less of the migraine type; at a given period the headache became transformed into a localized occipital pain, very different from that of the former attacks.

In Case II., the pain extended down the cervical spine, and was so much aggravated by movement as to suggest a rigid state of the neck. In Case I., there was once stiffness of the neck in an attack.

This peculiar headache was distinctly paroxysmal, but not at all periodical or influenced by any apparent outward circumstance. In both cases nausea accompanied the headaches, and in Case II. it is clearly stated that the nausea was secondary in point of time.

Case I. was made relatively clearer by the previous history of convulsions, and by the fact (not stated in the notes, but quite clear in my recollection) that the surgeons in attendance then (in 1876) found albumen in the urine.

Case II. was greatly complicated by the presence of symptoms of slight paralysis, partial ptosis and a weak right leg. I am now disposed to think that these phenomena, together with the astonishing debility, staggering gait, and the sluggish state of the mind which I observed in this patient, were due to brominism; a condition to which I have called attention as a possible serious complication in the diagnosis of disease.

I would also remark that the symptoms of renal disease were not marked; in one case there was no œdema, in the other a mere trace; neither patient had the dyspeptic symptoms or the frontal headache which often suggest renal disease, and neither patient has the "Brighty look" which is so well known.

It is to be observed that the occipital sensation in these cases was true pain, not the painful paræsthesiæ which are sometimes due to lithæmia and oxaluria, and sometimes to eye-strain, and which are erroneously (or rather insufficiently) designated as cerebral hyperæmia.

In some respects the story of these cases is imperfect, and I particularly regret the lack of observations upon the quantity of urine passed, and upon the state of the arterial tension.

Still I am inclined to believe that the publication of these cases may serve to render more accurate the diagnosis of occipital headache, and to illustrate the utility of critically examining the urine in cases of any degree of obscurity; more especially as occipital headache is scarcely mentioned as a symptom of uræmia.

THE LOCALIZATION OF DISEASES IN THE SPINAL CORD.*

I HAVE designed this lecture as a pendant to Dr. Gray's discourse upon the new anatomy of the spinal cord and its intracranial expansion. In the last fifteen years advances in the normal and pathological anatomy and the physiology of the nervous centres have progressed hand in hand, one illustrating and confirming the other.

At the beginning of this century, and from that time until some fifteen years ago, though great progress was achieved in the clinical description of spinal diseases, from Ollivier to Brown-Séquard; and though we have acquired some knowledge of the pathological anatomy of the points involved, almost

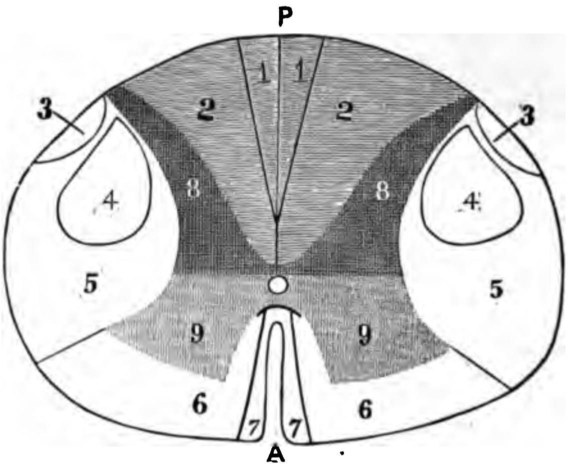

FIG. 1.

DIAGRAM OF TRANSVERSE SECTION OF THE SPINAL CORD.

A. Anterior median fissure. P. Posterior median septum. 1. Columns of Goll. 2. Columns of Burdach. 3. Direct cerebellar fasciculi. 4. Crossed pyramidal columns. 5. Lateral columns. 6. Anterior columns. 7. Direct pyramidal columns. 8. Posterior gray horns. 9. Anterior gray horns. Stippled part—Gray matter. Shaded part—Æsthesodic system. Unshaded part—Kinesodic system.

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* Delivered before the Anatomical and Surgical Society of Brooklyn, April 12th, 1880. Reprinted from the *Annals of the Anatomical and Surgical Society*, Brooklyn, vol. ii., No. 12, 1880.

no attempt had been make to localize the lesions of various diseases in definite sections of the spinal cord. The admirable spinal localization of the present day we owe to the accumulated labors of Cruveilhier, Türck, Charcot, Leyden and Erb.

I shall offer you to-night a brief review of this localization of diseases in the spinal cord, as founded upon normal anatomy, physiology and pathological anatomy. Such a study will be facilitated by admitting as practically correct a somewhat rough division of the spinal cord into two unequal regions, one including the posterior columns and the posterior gray matter, serving for the transmission of centripetal or sensory impulses, and the second, much larger, including the anterior gray horns, the anterior columns and the antero-lateral columns, serving for the transmission of motor centrifugal impulses. The former region is the æsthesodic system of the spinal cord, the latter the kinesodic system. The accompanying wood-cut illustrates the limits of these two systems, on a transverse section of the spinal cord.

The following table embodies the principal localized lesions of the spinal cord which give rise to definite symptom groups, and which we are able to diagnosticate :

LOCALIZED SPINAL DISEASES.

A. Systematic diseases of the spinal cord :

1. Diseases of the æsthesodic system—
    - *a.* Sclerosis of the posterior columns (progressive locomotor ataxia).
    - *b.* Ascending degeneration.
2. Diseases of the kinesodic system—
    - *a.* Degeneration of anterior ganglion cells (progressive muscular atrophy).
    - *b.* Inflammation of anterior gray horns (atrophic spinal paralysis of adults and children).
    - *c.* Sclerosis of the lateral columns (tetanoid paraplegia).
    - *d.* Descending degeneration,
        - *α.*—of spinal origin ;
        - *β.*—of cerebral origin.

B. Focal affections of the spinal cord (injuries, tumors, foci of softening, myelitis transversa, clots, etc.):

a. In lumbar enlargement.
b. In lower dorsal region.
c. In upper dorsal region.
d. In lower cervical enlargement.
e. In upper cervical region.
f. At the cauda equina.

It will be impossible to do more this evening than to consider the first of the two great divisions—viz., that of systematic lesions.

1. Diseases of the æsthesodic system.

At the present time there is only one disease of this class —viz., posterior spinal sclerosis or progressive locomotor ataxia. The lesion consists, roughly speaking, in sclerosis of the posterior columns of the spinal cord. Since 1873 closer analysis at the hands of Charcot and Pierret has shown that the primary and essential sclerosis occupies only the external part of these columns—that which is adjacent to the posterior gray horns, and which histology teaches us is traversed by fibres of the posterior roots on their way to the gray matter of the cord. In most cases which come to the post-mortem table the median fasciculi of the posterior columns, or the columns of Goll, are likewise sclerosed, but this alteration is to be looked upon as secondary, as a form of ascending degeneration, and probably as wholly disconnected with the symptoms. Almost always sclerosis of the external part of the posterior columns, or columns of Burdach, commences in the lumbar enlargement and advances upward. Ultimately, in old cases, the whole of the posterior columns as high as the medulla oblongata is sclerosed and degenerated, atrophied and hardened. Another part of the lesion of locomotor ataxia is a sclerosis and degenerative change in the posterior roots, extending to the ganglia on these roots and even involving them. In uncomplicated cases of locomotor ataxia, the anterior and antero-lateral columns, and the whole of the gray matter of the spinal cord, are healthy. The symptoms of the disease are chiefly of a sensory sort, and there is never any true paralysis in uncomplicated cases. The chief symptom in some respects, the initial symptom in the vast majority of cases, is a peculiar, almost pathognomonic neuralgia, which usually has its seat in the lower extremities alone, and sometimes affects the upper extremities and trunk, very rarely the head. The diagnosis of locomotor ataxia depends so much upon this symptom and its exact appreciation that I need no other apology for stating its characteristics with some fullness.

a. The pains are vagrant; they occur in innumerable spots in the affected parts, so much so that patients who have long had them are unable to enumerate the localities in which they have suffered; or, rather, they can hardly name a region which has escaped.

b. The pains do not occur in the course or distribution of recognized nerve trunks and filaments; they are local pains, and this peculiarity may serve (with *a*) to distinguish between the pain of sclerosis and the true neuralgia (sciatica, etc.).

c. The seat of the pain is commonly in an area of skin varying in size from that of a pea to that of a small hand. In many cases pains are referred to the muscles, to the vicinity of the bones, and even to articulations and viscera.

d. The pains are paroxysmal in a completely irregular manner; they may occur every few moments for hours in one spot, or be altogether wanting for weeks; or at times a single pain in a given region is the signal that the disease is not cured. It seems probable that the atmospheric disturbance (low barometer) which precedes a storm causes an increase in this symptom, or even calls it forth.

e. The pains are sudden, and vary in severity from the sensation caused by the penetration of a small knife-blade to that we may imagine to result from tearing through the tissues with a hook or large knife; or, the sensation is like an electric pain in suddenness. Perhaps most of the suffering in such cases is in the shape of stabbing pains in an ovoid or round area of skin (foot, thigh, arm, or shin) repeated every few seconds for hours, or even a whole day. The pain is often such as to make the strongest-willed man writhe and shriek. The seat of pain becomes hyperalgesic—*i. e.*, painful to the lightest touch; yet firm pressure may give relief. From their suddenness and electric character the pains of locomotor ataxia are often called fulgurating, or terebrating.

The *rationale* or physiology of these characteristic pains is found in the morbid anatomy. As described above, there is in this disease a sclerosis of the connective tissue in the posterior columns of the spinal cord, and chiefly in their lateral portions—*i. e.*, those portions which are traversed by fibres of the posterior roots. Irritation of these sensory nerve fibres is produced by the advancing sclerosis, and probably varies in degree according to changes in the circulation in the diseased spinal cord.

It should be borne in mind that fulgurating pains usually precede ataxia by three or four years; but this neuralgic period may be shorter (a few weeks) or much longer, perhaps indefinite (in a case of my own, twenty-nine years).

Other sensory symptoms of posterior spinal sclerosis are numbness and anæsthesia. The numbness often goes hand in hand with the pains, and is probably due to the same cause—viz., irritation of the posterior roots and their intra-spinal expansion. It usually affects the feet first, and seems to ascend. When the upper extremities are involved, the numbness first shows itself in the finger tips.

Anæsthesia is present in a large majority of cases; probably in all which enter the second period—viz., the ataxic period. It may progress to such an extent as to make the patient perfectly unaware of any excitation applied to the legs (or hands); and to render him ignorant of the positions and existence of the limbs without the aid of sight or touch, the patient "loses his legs in bed." The anæsthesia is caused by the actual destruction or great compression of the sensory fibres already referred to.

Destruction of the myeline and pressure on the axis cylinders by the sclerosed interfibrillar tissue may also explain a singular symptom—viz., the retardation of sensations—*i. e.*, the occurrence of a measurable time (10 to 120 seconds) between the pricking of a part and the acknowledgment of the pain by the patient.

Other symptoms of sclerosis of the posterior columns are motor to all appearances, yet in reality depend upon interference of the disease with the spinal sensory apparatus.

First. Diminished reflexes, pupillary, cutaneous, tendinous and visceral. For example, in many cases the pupils are small (may be unequal), and do not appreciably respond to light and shade, though they do change under accommodative efforts.

Again, if we tap the ligamentum patellæ in a patient suffering from fulgurating pains, or in one who has entered the ataxic stage, we observe that the quadriceps extensor femoris does not contract and cause a movement of the leg (the knee being semi-bent) as in health. This is known technically as absence of the patellar reflex, a new and most important symptom of posterior spinal sclerosis, one which in my experience hardly ever fails, and which deserves to be ranked as at least equal in importance with the fulgurating pains. Reflexes from cutaneous surfaces are also diminished or lost. The visceral reflex actions by which we micturate, defecate, and produce the sexual orgasm are likewise progressively impaired; and thus we find these patients impotent as a rule, and presenting constipation and slow, imperfect micturition as symptoms.

This reduction in reflexes is caused by disease of the intra-spinal sensory parts connected with the various organs and parts we test; the arc for reflex actions is impaired in the posterior columns of the spinal cord or in corresponding regions of the spinal tract.

The ataxic movement of the legs (and of the arms in some cases) which are characteristic of the second stage of the disease, appears at a variable period after the beginning of the neuralgic stage (three months to ten years or more) and is essentially characterized by an irregular, asynergic action of the muscular groups which serve to produce a given movement. The legs are jerked forward and outward, and the heel brought forcibly down in the attempt to walk; the fingers and arms oscillate and perform unnecessary excursions in trying to reach a given point, or accomplish a given action. Later, the irregularity of movement is so great that the patient is confined to bed.

We are not yet agreed upon a theory of ataxia, but these hypotheses are deserving of consideration:

a. That interference with sensory intra-spinal tracts diminishes the muscular tonus (chiefly produced in an unconscious reflex way), and that this atony varying in different muscles gives rise to the inharmonious movements.

b. The anæsthesia affects the muscles as well as the superficial parts, and thus diminution or loss of the "muscular sense" is caused, and the patients can no longer guide their contractions.

c. The sclerosis of the posterior columns affects other fibres beside common sensory ones—viz., those arciform or longitudinal commissural fibres described by Lockhart Clarke, and which seem to unite, for the purpose of harmonious action, the spinal centres for the various muscles of a group, or of a limb. If these commissural fibres be destroyed by sclerosis, we obtain ill-combined, asynergic muscular movements in the attempt to step.

There are many other symptoms in the course of posterior spinal sclerosis, but those just analyzed are the characteristic ones—the ones which are logically related to the lesion.

Under the head of diseases of the æsthesodic region we must also place the lesion of the posterior columns known as ascending degeneration. This lesion is limited to the posterior median columns, or columns of Goll, and diminishes in extent the higher we examine the spinal cord. We find this ascending degeneration above a spot in the spinal cord where it is compressed or destroyed, and it is also met with in progressive locomotor ataxia. Prof. Charcot and others teach that this extension of sclerosis from the columns of Burdach to the columns of Goll is a non-essential feature of locomotor ataxia, and is secondary. At any rate sclerosis or degeneration of the posterior median columns alone does not, as far as we know to-day, give rise to any special symptoms; hence I can give you no clinical picture to accompany the pathological statement. We know only that the lesion exists in a living patient by learning the pathological state of the cord from other symptoms present.

2. Diseases of the kinesodic system, including the anterior gray horns, the anterior columns, the antero-lateral columns, and the postero-lateral columns (or crossed pyramidal fasciculi).

There are, as indicated in the table, several very definite and distinct affections of those parts.

a. Degeneration of ganglion cells in the anterior horns. In this disease the protoplasm of these cells is very slowly transformed into granulo-fatty material, the cell processes are broken off, the nucleus and nucleolus destroyed, and in later stages there remains of the cell only a small mass of granules, or its place in the tissues may be vacant. These changes take place with extreme slowness, and the symptoms are correspondingly gradual in their appearance. For reasons which we do not understand, this granular degeneration may be limited to a few groups of ganglionic cells, or may involve a large number of the cells from the medulla oblongata to the end of the cord. We are not acquainted with the lesion above the medulla, although there is no reason why the nuclei of the fifth, sixth, seventh, fourth and third cranial nerves should not suffer. I wish to lay particular stress upon the slowness of the degeneration, because of its harmony with symptoms and its contrast with allied states.

Degeneration of anterior ganglion cells is externally expressed by muscular atrophy—usually progressive muscular atrophy. This singular disease shows itself often first in peripheral muscles, in hands and feet; in other cases it attacks the shoulder, arm and thigh muscles. Indeed, it may commence in any muscular group. Yet there are certain characteristic peculiarities in this wasting.

1. It is very gradual; fasciculi after fasciculi in the muscles undergo a diminution in size, lose their faradic contractility, and disappear, while adjacent fasciculi remain normal. There is no *paralysis* of a muscle, but a partial and gradual death of its constituents.

2. The atrophy affects, in the vast majority of cases, symmetrical and homologous parts. For example, both shoulders may be wasted, or the arms and thighs, or the forearms and legs. Unilateral muscular wasting is presumably not progressive muscular atrophy.

3. The muscles which are undergoing the early changes of this wasting are the seat of what are called fibrillary contractions. These are produced by the involuntary rapid contraction of fasciculi of muscular fibres in a muscle. Sometimes a patient is covered with them. Some years ago these fibrillary contractions were held to be pathognomonic, but I can assure you that this is not so, as they may be observed in lead palsy, in conditions of neurasthenia, in simple paralysis. Indeed, many years ago, Prof. Schiff, now of Geneva, showed that muscles separated from their motor nerves were prone to show fibrillary contractions.

The electrical reactions are diminished, but not altered in quality; and the reduction or loss of reaction (to faradism) is in direct proportion to the wasting. In partly atrophied muscles some fasciculi look well and contract well, while their neighbors are thin, and rise but feebly under faradism.

Uncomplicated degeneration of ganglion cells is unaccompanied by numbness, anæsthesia, or vesical symptoms, though aching pains may be present in the affected limbs.

b. Inflammation of the anterior gray horns; myelitis anterior in adults and in children.

Autopsies have shown that the lesion in this disease involves the anterior and central gray matter of the spinal cord, and that there may also be present, as secondary conditions, atrophy and degeneration of the antero-lateral columns (not distributed as in regular descending degeneration). The ganglion cells of the affected anterior horns are found in various states of change—swollen, containing vacuoles, filled with granulo-fatty matter, or shrunken and almost destroyed. Besides, the neuroglia round about these cells is always more or less altered. In some cases the lesion might be designated as a diffused central myelitis with destruction of anterior ganglion cells. It need hardly be added that there exists an absolute anatomical relation between the level at which the changes occur in the spinal cord and the distribution of symptoms at the periphery.

The symptoms of myelitis anterior are chiefly motor, very much as in group (*a*); but the morbid process being comparatively or absolutely rapid, they appear in a strikingly acute or sub-acute form.

First. Paralysis.—In some few cases this appears with almost apoplectic rapidity (suggesting hemorrhage in the cord); in the majority the paralytic phenomena are complete in one or two days. Often we learn from the mother of a child who has myelitis anterior (infantile spinal paralysis) that she put the child to bed well, or simply a little feverish, and that the next morning both legs, or one extremity, or all the limbs were limp and paralyzed. In a minority of cases (adults) the muscular groups in the limbs lose power slowly, and a week or several weeks elapse before the patient is obliged to lie abed. It is important to contrast this paralytic loss of power, affecting a whole limb, or a whole muscular group, with the atrophy described under the head of "Progressive Muscular Atrophy."

Second. Atrophy of muscles.—The palsied muscles in myelitis anterior always undergo, in the course of the first four or five weeks, marked and even extreme atrophy. This wasting, even in the most sub-acute forms of myelitis anterior, is rapid as compared with the most rapid forms of progressive muscular atrophy. Besides, in the former disease, the atrophy, like the paralysis, always affects a whole muscle, or a muscular group, or the muscular apparatus of one or more limbs, *en masse*, and is not, as in the latter affection, fascicular in distribution. Fibrillary contractions in wasting muscles are common in progressive muscular atrophy, and very rare in myelitis anterior.

Third. Remarkably distinct evidences of the degenerative reaction to electricity are obtained from the second to the tenth week. The nerve-trunks supplying the paralyzed muscular groups lose their excitability to faradism and galvanism, and these wasting muscles react only to galvanism, and that their reaction formula is altered from the normal: in general terms, we may say that $\text{An c c} = \text{Ka c c}$, or even $\text{An c c} > \text{Ka c c}$; and all contractions are slow and wave-like.

Fourth. The distribution of the paralysis is important for diagnosis and prognosis. In myelitis anterior the bladder and rectum are never paralyzed, and it is exceedingly rare to observe paralysis of the respiratory muscles, and of those which serve for deglutition. On the other hand, muscles supplied by the cranial nerves may be paralyzed.

Fifth. An important symptom not usually referred to is abolition of reflexes in the paralyzed parts, especially the tendon reflexes. For example, if the muscles of the thigh (quadriceps extensor femoris) be paralyzed, no patellar tendon reflex can be obtained, just as in posterior spinal sclerosis. The mechanism is in both affections the same, but in either case different portions of the spinal arc serving for reflexes are injured; in posterior spinal sclerosis the centripetal (sensory) portion of the arc is destroyed; in myelitis the anterior (motor) portion. At an early stage of myelitis anterior, and in cases (infantile) where much fat serves to obscure atrophy, this negative symptom acquires great value.

Sixth. Sensory symptoms are very slightly developed in myelitis anterior. In some cases a degree of numbness or other paræsthesia is experienced in the paralyzed limb for a few hours or days. In others severe neural pains are experienced. I style these neural because they apparently are in the course of nerve trunks or large branches, and are not in spots or patches like the fulgurating pains of posterior spinal sclerosis. Although the suffering in some few cases of myelitis anterior may be very severe, yet it is only careless observers who could mistake these pains for those of locomotor ataxia. Anæsthesia is never present in myelitis anterior. When it is observed in any marked degree, the case should be designated as diffused central myelitis.

Seventh. Fever is present in a majority of cases in children and adults. Other peculiar symptoms are vomiting, œdema of the extremities, hyperæsthesia of affected parts, delirium. These rare symptoms occur in the first stage of the disease. In the atrophic stage the patient's general health is usually good.

c. Sclerosis of the lateral columns has been recently described by Erb (1875) and Charcot (1876) as probably an independent or protopathic lesion of the spinal cord. To the symptom-group characteristic of this lesion, Prof. Charcot has given the name of spasmodic tabes, Prof. Erb that of spastic spinal paralysis. I have proposed the name of tetanoid paralysis or paraplegia, which seems to me most expressive.

The pathological anatomy of this affection is not as yet well established; it is doubtful if the lateral columns generally are sclerosed, or whether the lesion is limited to their posterior portions, as in descending degeneration (*vide infra*). The lesion is always (?) bi-lateral. Another uncertain point is whether the lateral sclerosis is primary or secondary—*i. e.*, due to a focus of disease centrally placed above the beginning of the sclerosis. It seems to me that the weight of evidence is in favor of the latter view.

The symptoms of tetanoid paraplegia consist in slowly increasing paresis in the legs (and arms rarely) with tendency to contracture and increase in all reflexes. The loss of power is exceedingly slow. The patellar tendon reflex is increased very early in the disease, and becomes greatly exaggerated. The skin of the feet forms the starting point of reflex muscular contractions when the patient is awake. Except at the close of the disease the affected limbs are relaxed in sleep. As a result of this increase of reflex action, there is a peculiar attitude and gait. The legs tend to cross one another, or actually do so from over-action of the adductors. The heel is drawn up, or at any rate raised, by over-action of the gastrocnemii and solei; the legs in stepping are stiffened, their muscles sensibly hardened. The extremities are in a tetanoid condition. In later stages, in bed-ridden patients, the legs may be fixed in semi-flexion and adduction. These symptoms indicate clearly that the proper spinal activity—its reflex and automatic activity—is increased.

Another motor disturbance, due to increased reflex action, is precipitate micturition and defecation. The patient must hurry, as from increased reflex action the bladder and rectum are apt to contract suddenly and involuntarily upon their contents. We have here an example of incontinence through spasm—a condition to be carefully distinguished from paralytic incontinence.

The muscles in parts affected with tetanoid paralysis retain their volume, nutrition, and normal electrical reactions.

Sensory symptoms are practically wanting in this disease; there is no anæsthesia, and seldom numbness.

If there were time I should like to draw a contrasting picture between this condition and sclerosis of the posterior columns; you can, however, easily do it for yourselves.

In little children we not very rarely meet with the symptoms of tetanoid paralysis involving the legs alone, or all the limbs. The child cannot stand or walk because of the spasmodic condition of the legs and the apparent absence of cerebral stimulus; no anæsthesia is present. In some cases we find microcephaly and idiocy conjoined. Prof. Erb was the first to describe these infantile forms (1877), and he is disposed to think that pathological researches will show as lesions, sclerosis or want of proper development in the postero-lateral columns. I incline to the view that in these tetanized children the entire motor tract, from its cortical (cerebral) starting point to its distribution in the spinal cord, is more or less incompletely developed. The operation of circumcision is still gravely advocated as a cure for infantile tetanoid paralysis, but we have no recent and uncontradicted statements of cures; besides, in my experience, Jewish children fully circumcised from birth frequently come to my clinic with these symptoms.

A combination of *b* and *c* is recognized under the name of amyotrophic lateral sclerosis (Charcot).

In this affection the lesions proper to *b* and *c* respectively are found in the spinal cord; viz., destruction of the anterior ganglion cells, and sclerosis (degeneration probably) of the postero-lateral (or crossed pyramidal) columns.

The symptoms correspond. We have a paralysis with atrophy in the upper and lower extremities, followed by contractions, with increased reflex in the non-atrophied and non-paralyzed muscles.

d. Descending degeneration: *α.* Of spinal origin. This takes place when the physiological continuity of the spinal cord is interrupted. For example, after division of the cord with a knife, after great pressure from a tumor, or from a displaced vertebra, or from a focus of transversely localized myelitis, we observe these changes. They differ completely above and below the seat of pressure or of section. Above the lesion we find an alteration almost limited to the posterior median columns (columns of Goll) extending indefinitely upward toward the apex of the fourth ventricle. The degenerated tissue appears in transverse sections as a wedge-like area, with its base resting on the pia mater and its apex not quite reaching the posterior commissure (see Fig. 1). Careful examination often shows on each side of the cord another tract of altered nerve tissue—viz., a portion of the postero-lateral columns lying posteriorly to the crossed pyramidal column, and extending to the pia mater and the posterior gray horn—the so-called ascending cerebellar fasciculus (Flechsig).

Below the lesion dividing the spinal cord the degenerations are found chiefly in two locations. First, in the crossed pyramidal columns (see Fig. 1); and, second, in the anterior columns, or, physiologically, the direct pyramidal columns. In these columns the descending degeneration extends to the lowermost portion of the cord. A more complete description of these columns will be given in the next section.

The symptoms of the ascending degenerative changes, if there be any, are now unknown. As regards descending changes, they very probably are expressed by a more or less defined tetanoid state of the paralyzed limbs. In some cases this is well-marked, and constitutes the spinal epilepsy of Brown-Séquard, or may resemble the symptom group (c) attributed to the primary (?) lateral sclerosis. No sensory symptoms accompanying either ascending or descending degeneration.

β. Descending degeneration of cerebral origin.

In this condition the foci of disease are found above the decussation of the anterior pyramids; by far the greater number within the cerebral hemispheres. The nature of the lesion is, of course, immaterial, providing it be a destructive one. Such, for example, are clots in the motor region of the brain (more especially involving the anterior two-thirds of the internal capsule), patches of softening involving the motor convolutions or tumors in similar locations. Usually such a lesion is unilateral, but once in a while bi-lateral lesions exist, and we must be prepared for the existence of bi-lateral symptoms.

From the focus of disease in these motor convolutions, or in the associated fasciculi of the corona radiata, the internal capsule (directly or indirectly involved), in general terms from the central part of the motor tract described to you by Dr. Gray, we can trace the degenerative changes with the naked eye (after six or eight weeks) to near the end of the spinal cord, through certain well-known columns or fasciculi of the cerebro-spinal apparatus. Let us take an example: A patch of softening involving the motor convolutions bounding the fissure of Rolando in the right hemisphere, now known to be the source of direct innervation for the left upper and lower extremities. Beneath the patch there is an altered bundle of fibres of the corona radiata, extending downward in the anterior (or middle) portion of the internal capsule; thence into the right crus cerebri, the right inferior half of the pons, the right anterior pyramid of the medulla oblongata. At the base of the brain, in the crus, pons, and medulla, the degeneration is superficially evidenced by an atrophy of the parts and color. Below the medulla oblongata the changes are to a considerable degree invisible to the naked eye, but appear upon transverse sections of the spinal cord. On examining such sections we find changes in different localities in the two halves of the spinal cord. In the immense majority of subjects, the evident, the principal change, or secondary degeneration, is found in the *left* posterior lateral column, or, more strictly speaking, the crossed pyramidal column, so called because it is the continuation across the median line of the anterior pyramid which has (partially) decussated at the point known as the pyramidal decussation. The exact location of this bundle (in the transverse section) varies somewhat at different levels in the spinal cord. In the upper cervical region it lies deep in the lateral columns, and as we examine sections made lower down we find it more posteriorly and laterally. It is always in the posterior-lateral column, to use a rough anatomical term, and never quite reaches to the periphery of the cord; it diminishes in size (area) from above downward, and is quite lost to the naked eye in the lower lumbar enlargement. In this crossed pyramidal column (see Fig. 1), I repeat, the principal descending degeneration is found. The other, smaller descending lesion, in our supposed case, is found in the right half of the spinal cord—*i. e.*, on the same side as the original disease and the intra-cranial degeneration-paths. This second spinal degeneration involves the anterior column of the cord, strictly speaking—*i. e.*, that fasciculus which lies between the anterior median fissure and the anterior gray horn—the so-called column of Türck (see Fig. 1). This column embraces those fibres of the anterior pyramid which have not crossed the median line; hence the better name for this fasciculus is the direct pyramidal column. In our supposed case we find the right anterior column more or less altered throughout the length of the cord.

To sum up: Below the pyramidal decussation the secondary degeneration is found both in the crossed and the direct columns derived from one of the pyramids—the right in our hypothetical case.

A most important law, discovered and abundantly proved by Prof. Flechsig, of Leipzig, is that of *variability in the pyramidal decussation.* He found that in most of his specimens the crossing took place as above described, the great majority of pyramidal fibres from the anterior pyramid crossing the median line and going to form the crossed pyramidal column, while a smaller number remained uncrossed and constituted the direct pyramidal column. But in one of his fœtuses this was reversed—*i. e.*, the direct pyramidal column was much larger than the crossed column. In several instances he found a degree of equality between the two columns derived from a pyramid. He concludes—and I entirely agree with him—that we must look upon the old law of decussation of motor fibres as liable to rare but remarkable variations. One result of such reversal of the law of decussation in a given individual would be to cause the paralysis from a cerebral lesion to appear on the same side of the median line. Although Brown-Séquard has written on the subject of paralysis from cerebral disease since the publication of Flechsig's great work (1876), he ignores these facts, which are alone sufficient to maintain the accepted hypothesis of cerebral motor action, and also to completely refute his iconoclastic theories, since if we admit that sometimes the pyramidal distribution is chiefly direct, we must deny the value of Brown-Séquard's three hundred cases of paralysis on the same side as the cerebral lesion. As long ago as January, 1878, I made use of this argument, which to my mind is convincing.

To return to the subject of descending degeneration:

Its symptoms consist in increased reflexes (from tendons especially) and contracture, in the wholly or partially paralyzed parts; arm and leg—rarely the face. It is useless to further describe this condition which we all see so often in uncured cases of hemiplegia. I would, however, call your attention to one important fact which I believe I was the first to notice and draw a conclusion from—viz., that during sleep in a warm bed the affected limbs are wholly or partially relaxed, and contract at once upon waking or upon exposure to the cooler air. I need hardly add that the contracture, like the paralysis in hemiplegia, is greatest in peripheral parts.

The classic theory of the physiology of contracture in hemiplegia is that it is due to the secondary degeneration—*i. e.*, actively caused by the lesion of the postero-lateral column. Seven years ago (see Archives of Scientific and Practical Medicine, Vol. I., p. 106, 1873) I rejected this hypothesis, and suggested a different one, which I have since elaborated and taught in my clinical lectures at the College of Physicians and Surgeons, New York. This hypothesis, which I intend shortly to publish in detail, is briefly that the spasm is due, not to direct irritation from the sclerosed (?) tissue in the postero-lateral column, but to the cutting off of the cerebral influence by the primary lesion, and the consequent preponderance of the proper or automatic spinal action—an action which is mainly reflex. This theory explains the phenomena observed in cases of primary spinal diseases with descending degeneration (α) and can be reconciled with results of experiments on animals (increased reflex power of spinal cord after a section high up, Brown-Séquard; inhibitory power of encephalon on spinal cord, Setschenow).

Want of time will prevent us from considering the focal affections of the spinal cord, classed in the table under section B.

A CASE OF ABSCESS OF THE LEFT FRONTAL LOBE OF THE CEREBRUM, WITH SPECIAL REFERENCE TO LOCALIZATION. *

On April 11, 1880, I was asked by Dr. J. Lewis Smith to see a case in consultation with himself and Dr. J. R. Leaming. The patient was a young married woman, aged about 28 years, who had formerly enjoyed good health and had borne several children. During the month of February one of these children had died after a severe illness, and she had undergone considerable fatigue. She seemed depressed, weak, and anæmic afterward.

About four weeks before the date of the consultation she complained of pain over the left eye. This was soon accompanied by swelling and exophthalmus, and on March 24th Dr. Knapp was called in and diagnosticated orbital (subperiosteal) abscess. This was opened on March 26th by Dr. Knapp.

It was remarked that the pus was under great tension, and that it spurted out a considerable distance when released. Pain ceased at once, the exophthalmus disappeared, and the wound quickly healed. During the first few days of April all seemed going on well; the wound was healed; the patient was free from pain; she was taking tonics, and on the 3d made a call on a near neighbor.

During the night of April 3d and 4th, one week before my examination, she awoke with severe headache and vomiting; ever since she has lain abed, presenting the following symptoms: headache, chiefly mastoid and through the base of the skull; occasional vomiting; irregular respiration; irregular and very slow pulse, varying from 60 to 50 beats per minute; stupor and general feebleness. As negative points there were no symptoms about the eyes, objective or subjective, except a partial ptosis of the left upper lid (which had been incised); no fever, chills, convulsions, paralysis, aphasia; at no time had there been coma. The urine was free from albumen.

Examination.—Patient was soporose, but could be aroused by loud speaking; she answered questions as if half asleep, but in such a way as to leave no doubt as to the preservation of language. She put up both hands to the mastoid regions when indicating the seat of pain. A minute inspection showed no paralysis except about the left eye, whose upper lid drooped and whose internal rectus was inert. The pupil on the left side was not fully dilated, but it was a little wider than the right. The optic nerves appeared somewhat congested, and were dim at their periphery, but there was no actual choking. Patient appeared to feel pinching well everywhere. The thermometer showed no fever. The pulse varied from 53 to 66 beats per minute, and it was a reluctant, delusively full pulse, with no real strength. The breathing was easy and regular, but friends of the patient described quite well a Cheyne-Stokes breathing which they had observed. There was neither redness nor tenderness about the site of the orbital abscess.

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* Reprinted from the *Archives of Medicine*, vol. v., No. 1, February, 1881.

I diagnosticated an abscess of the brain, probably in the left frontal lobe,

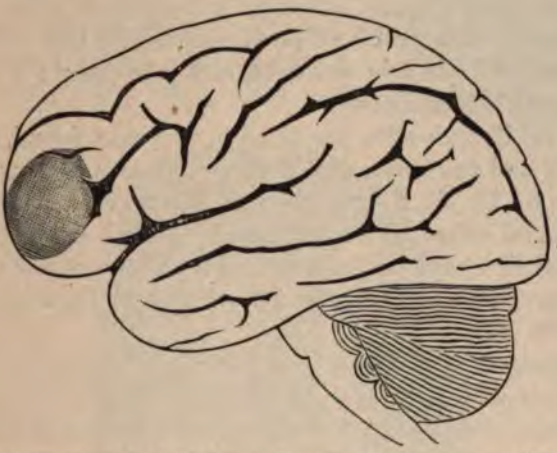

FIG. 1.—Apparent location of the abscess, drawn on an Ecker's diagram of the brain.

and expressed the opinion that the patient was in imminent danger. She died the next day in a comatose state, no new symptom having been observed.

It was then learned that for two years Mrs. F. had suffered from frequent attacks of headache, lasting several hours. The pain was frontal, and sometimes extended along the nose and into the left temple. There had never been symptoms of chronic nasal catarrh.

The autopsy was made on April 13th, about thirty hours *post-mortem*, in the presence of Drs. H. Knapp, J. R. Leaming, J. Lewis Smith (the attending physician) and Richard Wiener. We found a large abscess, the size of an English walnut, in the left frontal lobe. It seemed to lie wholly under the cortex cerebri, in the convolutions of the orbital lobule and in the second frontal convolution. Viewing the hemisphere from the side, the apparent posterior limit of the abscess was the anterior border of the lower part of the third frontal gyrus. Fig. 1 indicates the seat of the soft, fluctuating, bulging abscess. Its depth and penetration were not then determined, as it was thought best to harden the brain as a whole before making sections.

The external connections and origin of the abscess were most interesting. There was only one point of adherence between the diseased frontal lobe and the dura mater, and that was over the orbital plate of the frontal bone immediately under the swollen frontal lobe. There the dura mater was thickened and adherent to the pia mater and cortex cerebri, forming the inferior wall of the abscess, over a space as large as a ten-cent piece (about 15 mm.). Under this patch of pachymeningitis the orbital plate of the frontal bone was necrosed and perforated; a probe was easily passed into the orbit.

In the orbit, under its periosteum, pus was found, and parts of the roof and the inner wall of the orbit were carious. Careful dissection by Dr. H. Knapp showed disease of a similar kind in the ethmoidal cells and frontal sinus. I need say nothing more of the condition of these parts and of the pathology of the orbital abscess, as the case has been fully reported from this point of view by Dr. Knapp.*

The appearance of the necrosed orbital plate and of the thickened, adherent dura mater was precisely similar to what I have several times seen in cases of suppurative disease of the internal ear with cerebral abscess by contiguity. The genesis of the abscess must have been alike in the two situations.

In December, the brain having been sufficiently hardened in bichromate of potash solution, I imbedded it in Gudden's microtome, and made several horizontal sections through the whole brain with the view of demonstrating the relations of the abscess. These cuts showed that the abscess was of quite as large a size as at first supposed, almost perfectly globular in shape, measuring about 38 mm. in diameter. It contained ordinary pus, and was lined by a distinct membrane 1-2 mm. thick. The anterior, inferior and external limits of the abscess were thinned cortex and pia mater; superiorly, posteriorly, and internally, it was bounded by apparently normal white substance. The whole of the white centre of the frontal lobe, except a portion near the convexity of the hemisphere, was destroyed to within 10 mm. of the folds of the island of Reil, and about 8 mm. of the head of the nucleus caudatus. The mass of white substance connecting the inferior and posterior part of the third frontal convolution and the anterior gyri of the island of Reil with the internal capsule, was uninjured.

This last fact is of capital importance in estimating the bearing of this case upon the current notions of cerebral localization

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* *Archives of Ophthalmology*, vol. ix., p. 185, 1880.

The above description of the topography of the lesion, especially its posterior limitation, is made from the surface exposed by the lowest cut made, viz., one passing through the speech-centre of Broca, about 10 mm. above the apparent commencement of the fissure of Sylvius (pia still adherent). Fig. 2 is faithfully drawn from a photograph taken of this section-surface. The rest of the brain was healthy to the naked eye.

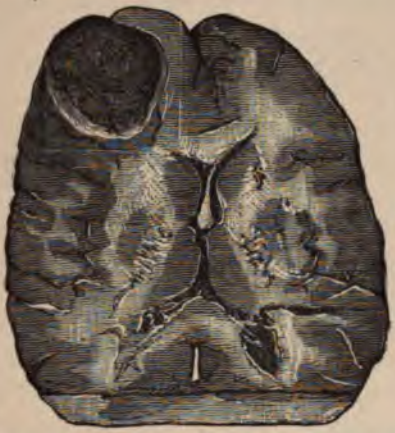

FIG. 2.—Relations of the abscess as shown in a horizontal section of the brain made at the level of Broca's speech-centre. Drawn from a photograph of the specimen. Occipital lobe cut off.

This remarkable case seems to me of much importance as a negative contribution to cerebral localization. It is in exact accord with recent experimental data, and with the *post-mortem* finding of the last ten years, that an abscess placed like this one should give rise to no motor symptoms, and should not cause aphasia. It is wholly within what are now called the inexcitable districts of the brain. The only symptoms present were the partial paralysis of the left third nerve (more immediately caused by the orbital abscess?) and signs of intracranial pressure. Yet it is important to note that in spite of the enormous pressure which must have existed there was no actual neuro-retinitis.

I have elsewhere reported another case of (smaller) abscess in precisely the same location (left frontal lobe) in which no symptoms referable to this lesion were present.*

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* A contribution to the study of localized cerebral lesions. Transactions of the American Neurological Association, vol. ii., pp. 122–4, N. Y., 1877.

On the other hand numerous autopsies are on record in which a smaller lesion (softening, hemorrhage, etc.), placed a centimetre farther back in the left frontal lobe, involving the posterior part of the third frontal gyrus or the band of white substance between it and the nucleus caudatus, has given rise to severe symptoms, hemiplegia or aphasia, singly or combined.

In the paper just quoted I have described such cases.

ON THE EARLY DIAGNOSIS OF SOME ORGANIC DISEASES OF THE NERVOUS SYSTEM.*

PROBABLY no one would deny the desirability and utility of making an accurate diagnosis of disease at the earliest possible period, and one of the results of recent progress in the medical art is increased possibility in this direction. We can recognize diseases which, though existing, were unknown to practitioners of thirty or fifty years ago, and we can also determine the existence of some of these affections at a much earlier period of their evolution than we could ten or even five years ago. The sciences of semeiology and of diagnosis have unquestionably progressed greatly in the last generation, and this is more especially shown in the history of specialties, as ophthalmology, dermatology, gynecology, and neurology. I may be permitted to say that it is a duty and privilege of the specialist to inform the profession at large of the advances made in his department in diagnosis and therapeutics, in order to enable the general practitioner to apply the new knowledge, or the confirmed old knowledge, to the advantage of his patients.

It is with such a motive that I would call your attention to the possibility and desirability of an early diagnosis of two or three organic diseases of the nervous system. Probably I shall name no new symptoms, but will aim to call your attention to the really valuable symptoms of these affections, and to the significant grouping of these symptoms.

I have selected three affections which are now quite well known to us, and yet which, judging from my experience, are frequently ignored during long periods of their formative stages: I refer to posterior spinal sclerosis (progressive locomotor ataxia), dementia paralytica, and cerebral tumor.

I. *Posterior spinal sclerosis, or progressive locomotor ataxia.*

While willing to admit the occasional occurrence of abnormal cases of this disease, in which ataxia appears with little premonition, yet I claim that the general practitioner at the present day should diagnosticate the disease in the clearly defined first stage, or pre-ataxic period, which may last from a few months to several years. The vast majority of cases exhibit this first stage, and its symptoms are peculiarly characteristic, if not pathognomonic. In general terms the symptoms of this first stage consist in peculiar pains, and in reduction or abolition of reflex movements in different parts of the body, and from a combination of these a diagnosis of great probability of accuracy can be made years before the patient's gait becomes disordered.

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* Reprinted from the *Medical Record*, Feb. 26, 1881

If we assume that nineteen out of twenty victims of posterior spinal sclerosis pass through this neuralgic or pre-ataxic stage, we will not be far out of the way.

The pains of posterior spinal sclerosis are almost pathognomonic, especially when described by an intelligent educated patient. They have the following characters:

a. The pains are vagrant; they occur in innumerable spots in the affected parts—so much so that patients who have long had them are unable to fully enumerate the localities in which they have suffered; or, rather, they can hardly name a region which has escaped.

b. The pains do not, as a rule, occur in the course or distribution of recognized nerve-trunks or branches; they are local pains, and this peculiarity may serve (with *a*) to distinguish between the pains of sclerosis and those of true neuralgia (sciatica, etc.).

c. The seat of pain is commonly in an area of skin varying in size from that of a pea to that of a small hand. In many cases pains are also referred to the muscles, to the vicinity of bones, and even to articulations and viscera.

d. The pains are paroxysmal in a completely irregular manner; they may occur every few moments for hours in one spot, or may be altogether wanting for weeks; or at times a single pain is the signal that the disease is not cured. It seems probable that the atmospheric disturbance which precedes a storm (areas of low barometer) causes an increase in this symptom, or even calls it forth.

e. The pains are sudden, and vary in severity from the sensation caused by the penetration of a small knife-blade to what we may imagine to result from tearing through the tissues with a hook or large knife; or the sensation is like a painful electric shock. Perhaps most of the pain in such cases is in the shape of stabbing pains in an ovoid or round area of the skin (foot, thigh, arm, shin, etc.), repeated every few seconds for hours or even a day or two. The suffering is often such as to make the strongest-willed man writhe and shriek. The description of the pains, *i. e.*, their comparison with known sensations or physical conditions, varies greatly, according to the fertility of the patient's imagination and his command of language. From their suddenness and electric character the pains of posterior sclerosis are often called fulgurating or terebrating. The seat of pain usually is hyperalgesic, *i. e.*, painful to the lightest touch during the paroxysm; yet firm pressure sometimes gives relief.

Second.—Diminution of various reflexes throughout the body.

This is best observed in the iris and at the patellar tendon, though the constipation and imperfect micturition which are such frequent symptoms of the disease are phenomena of the same order.

a. The impairment of iritic reflex action ("pupillary reflex") was first intelligently studied in 1869 by Dr. Argyll Robertson, of Edinburgh. His observations have since been abundantly verified by numerous observers, and an exhaustive paper on the subject has been published by Prof. W. Erb, of Leipzig, in the *Archives of Medicine*, October, 1880. Robertson and others after him noticed that the pupil of tabetic patients did not dilate in the shadow and contract in the light, as do normal pupils; and they further observed that during the effort of accommodation there occurred a normal pupillary contraction. In other words, the reflex iris movements were abolished, while its associated quasi-voluntary movements were preserved. These phenomena I have observed in almost all my patients suffering from posterior spinal sclerosis, and I am in the habit of calling the attention of students to the symptom. In two of the patients now under my care this condition is not present, but these have been cases of abnormal sclerosis, in which all the symptoms appeared in a most irregular manner.

The pupils in a suspected case of posterior spinal sclerosis are to be tested in the following manner: the patient is placed, seated or standing, facing a brightly illuminated window, and told to keep his look fixed on some distant object, such as a house or tree. By alternately closing and opening the lids, or better, by shading the eyes with one's hand momentarily, it is easy to see if the pupils change diameter. It is of the utmost importance that the patient's intelligent assistance be secured, in order that his gaze shall remain adjusted for distance. In a given case the absence of reaction to light having been noted, we next hold up one finger or a small object within a foot of the patient's face and bid him look at it. At once the pupils contract, and do so in proportion to the accommodative effort and the coincident convergence; when the patient looks at the distant object, and relatively or absolutely relaxes his accommodation, the pupils dilate again.

The finding of such a condition of the pupil—the existence of *Robertson pupils*, if you will allow the expression—is now considered of nearly as much importance for diagnosis as the occurrence of fulgurating pains.

b. Diminution and abolition of reflex action in the peripheral apparatuses is best studied at the knee.

We test the so-called patellar reflex, or knee reflex, or patellar tendon reflex in the following ways: the patient being seated, is told to cross one leg over the other in a natural manner, and to let the muscles relax; or seated, we place our left hand under the popliteal space, tell the patient not to help us, to let the leg hang loose, or, in popular parlance, "dead," and lift the whole limb so that the foot swings a couple of inches above the floor; then we tap the skin over the whole of the region from the insertion of the quadriceps femoris to the tuberosity of the tibia, with one or two finger-tips applied as in percussion. The place whence a reflex quadriceps contraction is most apt to occur is about midway between the lower end of the patella and the tibial protuberance. The taps should be gentle at first, and if these fail, harder ones are to be tried. A third mode of procedure, which is very good indeed, is to seat the patient on a table so that his legs dangle some two or three inches beyond its edge; then we tap the patellar region as above described, without supporting the thigh with our left hand. The test may be well done through the patient's clothing, yet it is desirable, especially in doubtful cases, to tap the bare skin. Another important precaution is to secure the absolute relaxation of the patient's muscles, and to divert his attention from what you are doing. Even with all precautions it is sometimes next to impossible to secure this indispensable muscular relaxation. In the healthy subject this test develops a contraction of the quadriceps extensor femoris and causes an extension of the leg, or a sudden jerk. In a very early stage of posterior spinal sclerosis no contraction takes place.

I would also call attention to the occasional occurrence of reflex movements of the thigh produced by contraction of the iliac group of muscles during the knee test. I have an example of this distant reflex action in a typical case of sclerosis of the posterior columns, in which the quadriceps does not contract at all.

While claiming very great diagnostic value for this negative symptom, I would not be understood as attaching pathognomonic significance to it, as we all know that there are a few seemingly healthy individuals in whom the patellar tendon reflex is lacking, and also that there are other diseases which diminish or abolish it. Indeed, I may say that I recognize no pathognomonic symptom, and even in attempts to push diagnosis to an extreme delicacy, would urge that reliance be placed on the grouping of symptoms, rather than on any one of the signs, however constant and important it may appear.

Physiologically analogous to this condition of loss of tendinous reflexes is the flabby state of the muscles in the affected parts. This is not due to any positive atrophy, as electrical tests show no marked departure from the normal reactions; but to impairment of what physiologists call muscular tonus, a state of partial contraction or tension of muscles which is kept up by the inevitable and continued excitation of the cutaneous nerves by air, clothing, surrounding objects, etc., acting in a reflex way through the spinal cord. It has been recently claimed that this loss of muscular tonus was the most important factor in the production of the ataxic movements which characterize the second stage of the disease.

The vesical and rectal reflexes are diminished in posterior spinal sclerosis. Slow, irregular micturition is complained of by most patients, in the first stage and in the second. We usually micturate without using much volition, but the tabetic patient is obliged to strain and to try hard to pass water. Defecation is, like micturition, a semi-voluntary act, and in the late first stage of the disease in question, constipation becomes more and more marked, and that through loss of the automatic or reflex action of the rectum and adjacent muscles.

The sexual act is, in my experience, frequently impaired and sometimes almost lost before the second stage sets in. The acts of erection and emission are usually brought about in a reflex manner by irritation of the skin and mucous membrane of the genitals. As a result of diminished spinal reflex action we have imperfect erections, and either premature emission, or, what is more common, I believe, very slow production of the orgasm, and impossibility of repetition within a reasonable time.

Some writers admit abnormally great sexual power in the early stage of tabes, but I am not sure to have met with more than one or two cases in which this seemed to be the case. In one of the patients, a female, I became convinced that her extraordinary capacity for sexual intercourse was not in a strict sense pathological or pre-tabetic, but had been marked in one shape or another from childhood.

It seems reasonable at the present time to advance this general proposition: that in posterior spinal sclerosis the various reflex actions performed by means of those portions of the cord which are the seat of sclerosis, are diminished or lost; or, to put it in another way more useful for practice, it may be said that the limitations of loss of reflex action in different parts of the body accurately indicate the limits of sclerosis in the posterior sensory apparatus in the spinal axis.

Third.—The occurrence of paralysis of ocular muscles.

A very large proportion of tabetic patients tell of past or present diplopia, and in a certain number of cases the ocular paralysis precedes the pains and ataxia by several years. So true is this statement, that it has become an established practice with neurologists and ophthalmologists to suspect posterior spinal sclerosis in adults who present themselves with strabismus, diplopia, or ptosis. In such a case we should carefully question the patient about the occurrence of fulgurating pains, and test the pupillary and tendinous reflexes. I need hardly add that another obligatory line of inquiry in such cases is with reference to symptoms of syphilis.

The same remarks apply to atrophy of the optic nerve, which is occasionally an early symptom.

I have not the time to refer to the gastric, laryngeal and rectal crises and the peculiar forms of arthritis which once in a while occur early in the disease.

It seems to me that, by a critical appreciation of the above symptoms in a patient, the diagnosis of the first, or neuralgic stage, of posterior spinal sclerosis is as certain as the diagnosis of any internal disease, not excepting such affections as pneumonia or valvular cardiac disease. Several autopsies are now on record, made during this first stage, and in these, sections of the cord showed sclerosis of the posterior columns. I have one such observation of my own: fulgurating pains for about thirty years, absence of patellar reflex while under observation (two years), dilatation of one pupil, no trace of ataxia. The sclerosis of the posterior columns in this patient's spinal cord is visible to the naked eye.

It is often objected that the pains of ataxia are not absolutely reliable for diagnosis. This may be true when the patient is stupid, or when the physician is not careful to ascertain the precise character of the pains.

The only two conditions in which pains somewhat resembling fulgurating pains occur, in my experience, are paralytic dementia and gout. In the former disease, slight fulgurating pains—"smaller" pains, if I may be allowed the expression—are described by the patients; but in many of these cases autopsy shows that, besides the cerebral lesions proper to the disease, the posterior columns of the cord exhibit pathological alterations; so that these cases are, after all, *quasi*-tabetic. The sharp pains of gout are short, stabbing pains in the skin of various parts of the body, compared by the patients to the prick of a needle, cold or hot. There is no tendency to repetition of the pain in one spot for hours or days; the sensations appear in various parts of the body, and are bearable. It is but right to add that this statement is based on very few observations, and requires verification.

The differential diagnosis of fulgurating pains from the pains of neuralgia, strictly speaking, is very easy. In neuralgia the pain is in the course and distribution of one or two (seldom) nerve trunks and their branches; it may be paroxysmal, but does not assume the excessive irregularity of the tabetic pains—agony for a few hours, and freedom from pains for hours, days, or weeks. The hyperæsthesia, in fulgurating pains, is at the seat of pain. In neuralgia we find regular "tender points" along the nerve trunk, or where its branches become superficial. The lightest touch causes pain in the painful districts in tabes, while the tenderness of nerves in neuralgia is usually demonstrable only by firm, localized pressure. Further, true neuralgia is seldom bilateral, while it is the rule for fulgurating pains to appear on both sides of the median line—in both lower extremities, for example. A last important distinction is that neuralgia is relievable or curable, whereas fulgurating pains are practically incurable, and are fully relieved only by morphia injections.

The confusion so often made between "rheumatism" and the first stage of sclerosis is even less pardonable. Of course no practitioner would mistake fulgurating pains for articular rheumatism; the error is with respect to "rheumatism," so-called, affecting muscular masses, and aponeuroses. In these affections the pains are usually dull, nearly constant, and distinctly aggravated by movements. Pressure must be firmly made upon the parts to produce pain, whereas in fulgurating pains the condition is one of cutaneous hyperalgesia under a slight touch. Again, this "rheumatic" condition is distinctly amenable to treatment (counter-irritants, etc.), whereas the pains of posterior spinal sclerosis are, in one sense, incurable.

II. The second disease of the nervous system to which I would direct your attention as the object of more exact and earlier diagnosis is *paralytic dementia.* By this term is meant the passive form of an affection which consists in peri-encephalitis, adhesion of the meninges, and various secondary, degenerative changes in the brain and in the posterior columns of the spinal cord. Chronic peri-encephalitis also presents itself in an active or delirious form, which is known as general paralysis or paresis. In neither form is there a positive condition of paralysis at any time, except as a complication from the occurrence of cerebral hemorrhage or softening. Both the semeiological names, paralytic dementia and general paresis, are, strictly speaking, misnomers; yet we accept them as sufficient.

The semeiology of peri-encephalitis is complicated, and it would be beyond the scope of this essay to describe it in detail. I merely wish to call your attention to the symptoms which, in my opinion, are earliest in their appearance and significant of an incurable disease. These are tremors or fibrillary contractions in various muscular groups, especially in the tongue, facial, and brachial muscles; a tremulous, thick, and vibratory speech; inequality of the pupils; dementia.

The tremor of paralytic dementia probably first makes its appearance in the facial and lingual muscles. It consists in non-rhythmical contractions of small muscles or of fasciculi of muscles, which are either present in the quiescent state of the features, or are excited by emotion or by the performance of a voluntary movement, as showing the tongue or the teeth. Sometimes innumerable fine, fibrillary tremors cover the face, while in some cases the movements are coarser and irregular enough to merit the term choreic. The tongue exhibits both sets of tremors—the very fine, fibrillary ones and the large, choreic oscillations. There is also, though usually at a later stage, some shrivelling or atrophy of the tongue.

The hands are tremulous, usually in a fine semi-rhythmical way. This trembling is sometimes scarcely visible, but is perceptible as a delicate parchment-like fremitus on holding up the patient's extended fingers between ours. In the lower extremities the tremulousness is not apparent.

The speech is affected as a result of this tremor and as the result of a certain want of co-ordination in the muscles of articulation. Words are quickly spoken, with some syllables omitted or blurred, or with a terminal syllable left off. The articulate sounds which are produced are heard as vibratory or tremulous, and the speech seems thick. Patients semi-unconsciously avoid long or difficult words in conversation, and even seek roundabout ways of expressing their meaning by shorter words. Besides this vibratory tremulousness in articulation there is an imperfection in the pronunciation of words, long words especially. Remedy is pronounced "remdy"; constitution, "constution"; infallibility, "infalliby." The last syllable may be badly sounded, or even omitted. I have known this characteristic speech to be the only well-marked symptom, and to be followed by dementia, exaltation, etc. Occasionally a patient comes to us complaining of this defective articulation. I now recall two such cases, one of which died three years later in a German private asylum, with all the symptoms of general paralysis.

Just as spoken language is affected by the facial and lingual tremor, so is the handwriting altered by fibrillary contractions in the muscles which govern the movements of the fingers. A tremulous, jagged, wholly irregular handwriting results, and in some cases, where dementia is present, words or syllables are frequently omitted in composition.

The pupils, in paralytic dementia, are either very small or irregular, usually the latter. The reaction of the iris to the influence of light may be diminished or abolished.

I may here say, by way of parenthesis, that small and unequal pupils in a person of middle age, from twenty-five to sixty, should lead to an inquiry into the possible existence of one of three morbid states, viz.: paralytic dementia (or general paralysis), sclerosis of the posterior columns, cardiac or aortic disease (intrathoracic disease).

In my experience, the patellar tendon-reflex is often increased in paralytic dementia.

The dementia, or failure of mental power, is sometimes impossible to detect until after the more peripheral physical symptoms have existed for some time. It is possible for the psychical symptoms to precede the physical; sometimes the two appear to develop simultaneously; usually, I believe, the physical symptoms already studied are apparent for months before the mind shows decay.

Dementia is evidenced by impairment of memory for recent events, by loss of the power of comparison, and consequently of judgment. Many of the automatic or quasi-automatic acts of every-day life which form a part of the patient's manner and individuality are badly performed or omitted. This leads to what is known as change of character in the subject; he becomes less neat in his attire or personal cleanliness; he loses his table-manners, handling his spoon, fork and knife awkwardly, soiling his clothing with drippings of food, etc. The impairment of judgment is probably one of the factors in the immorality and tendency to alcoholic indulgence which are so frequent in this disease.

Yet, in the midst of this increasing moral wreck, so visible to the immediate relatives of the patient, there may remain a degree of correctness in thought and success in every-day occupation which may impose upon strangers, and even upon a judge and jury. The things which the patient is in the habit of doing every day, and about which he has thought many years, such as professional work and business transactions, may be fairly well executed, while the tremors, pupillary irregularity, impaired articulation and handwriting, together with alteration of moral character, make the medical observer recognize a fatal progressive disease of the brain. These cases come more frequently under the observation of general practitioners than under that of the specialist, whether asylum physician or neurologist. They are very frequent in our midst, and their early recognition may save much disgrace and impoverishment to families, though, alas! it does not pave the way for more successful therapy.

I would repeat, that a person exhibiting tremors of the facial muscles of the tongue and hand, a vibratory and slurred speech, angular or tremulous handwriting, and irregular, small pupils, should be suspected of having chronic peri-encephalitis or paralytic dementia. The addition of gradual failure of mind—dementia—makes the diagnosis certain. In case there should be superadded exalted notions, with maniacal attacks and epileptiform seizures, the case deserves the name of general paresis; and as such is the form more usually seen and studied by asylum physicians.

It has been claimed in the last four years by Fournier and others that cerebral syphilis, in the shape of arteritis, partial arachnitis and localized peri-encephalitis, might give rise to the symptoms of paralytic dementia. I am in accord with Dr. Julius Mickle and others in believing that it is often possible to distinguish the idiopathic from the syphilitic dementia. The latter is, comparatively, much more acute (or rather less chronic), in its development; in it we do not observe the very fine muscular tremors as an early symptom; the pupillary disturbance consists usually of mydriasis of one side, with or without other signs of third-nerve palsy; the speech defect is a coarse thickness in pronunciation, rather than a vibratory, tremulous sound, which, when once heard, can never be forgotten. There are well-marked paralytic symptoms, usually hemiplegic, and decided epileptic phenomena in syphilitic cortical diseases. The dementia is seemingly more profound, causing an apparent imbecility with want of control over the sphincters. Altogether, the symptom group is much more threatening in appearance; yet great improvement, or even apparent cure, may be obtained in very bad cases by the use of mercury and by heroic dosing with iodide of potassium. This therapeutic proving of a disease is of course valuable in practice, but logically it cannot be termed a diagnosis, and it is a reproach to the present state of our science that in several types of disease we should be obliged to resort to it.

III. The third organic disease of the nervous system which should, it seems to me, sometimes be recognized with positiveness much earlier than it now is, is *tumor of the brain.*

In making this statement I am perfectly aware that some cerebral tumors produce no distinct or special symptoms during life, and that others produce incongruous and apparently paradoxical symptom groups. Some years ago, before the physiology of the brain was as well understood as it is now, we could offer no explanation of these perplexing cases, which seemed to destroy our rules of diagnosis. To-day we have acquired an approximately correct knowledge of which portions of the brain (cerebrum especially) are excitable and capable of causing symptoms, and which are inexcitable and may be the seat of extensive disease without clear indications. This I say without reference to the finer localization theories of the last five years. We know quite positively, for example, that extensive lesions may exist in the anterior and inferior portions of the frontal lobes, in the sphenoidal lobes, and in the occipital lobes of the cerebrum, and in one-half of the cerebellum, without causing any symptom specially useful for diagnosis, such as will be considered later on. We have also learned, from Flechsig's researches, that the decussation of the motor tract, just below the anterior pyramids of the medulla oblongata, is variable in amount, and that in some cases there may be no crossing of fibres, or hardly any. This important law of variability in the pyramidal decussation enables us to correctly appreciate the rare cases in which a cerebral lesion produces symptoms (paralysis or spasm) on the same side of the body as itself—cases which have been so urgently pressed upon the profession by Brown-Séquard in the last ten years as proofs that our physiological laws of cerebral action and of the productions of symptoms were all wrong. These laws stand to-day, I believe, only strengthened by the exceptions which have been adduced.

All I wish to say is, that tumors located in what we now term the excitable region of the cerebrum, or the motor zone, are capable of very early recognition.

The region which receives the name of motor zone is irregular in shape, and perhaps its limits are not yet well ascertained. In a general way we may say that it includes the median region of each hemisphere, in particular the posterior extremity of the third frontal convolution, the upper half of the second and first frontal, the ascending frontal and ascending parietal convolutions, the anterior gyri of the island of Reil, the paracentral lobule on the inner surface of the hemispheres, and, perhaps, a large part of the upper set of parietal convolutions. These are the motor convolutions, and embrace the so-called motor centres of Ferrier. Besides, we must include under the name of motor zone, or region, those fasciculi of white substance which connect the above-mentioned gyri with the crura cerebri, constituting the anterior half (or less) of the internal capsule as it passes between the nucleus lenticularis on the outer side and the nucleus caudatus and thalamus opticus on the inner side.

The succeeding remarks apply to tumors which involve any of this large expanse of cerebral substance, either in its external gray matter or in the fasciculi of white substance lying between the motor convolutions and the central gray bodies.

The symptoms which I think are characteristic of tumor in the motor zone of the hemispheres are :

Localized convulsions in peripheral muscles; equally localized paralysis of peripheral parts; neuro-retinitis or choked disk; localized headache. The symptoms are named in the order of their frequency and importance.

The initial convulsions of cerebral tumor are sometimes restricted to one side of the face, one hand, or even two fingers, or one leg. The spasm is usually tonico-clonic, but may be wholly clonic or jerky. In many cases this localized spasm is unaccompanied by loss of consciousness or vertigo, and it may remain localized in the part first affected during many attacks, extending over weeks and months of time. The patient feels the muscular contraction before it becomes evident, thus constituting a sort of aura. In some cases almost from the first, in nearly all cases after a while, the convulsion involves more muscles on one side of the body; it seems to ascend or descend, to use the patient's expressions, and there results a hemiplegic epileptic attack with loss of consciousness. Again, the attack may begin in a small peripheral part, involve the whole of one side of the body, and later affect the opposite side, thus constituting a full epileptic attack. The patient is able to watch the progress of the spasm for a number of seconds or minutes before losing consciousness, or being thrown down, and we may take advantage of this peculiarity to instruct the patient in the use of the tourniquet or bracelet, placed on the limb just above the seat of first spasm, to cut short the attack by pressure.

This distribution of spasm, and its possible occurrence without loss of consciousness, are signs which most positively distinguish these symptomatic convulsions from the ordinary epilepsy which we constantly encounter.

As early as 1827, a French physician, Bravais, described the hemiplegic form of epilepsy, and showed its relation to gross cerebral disease; but it is to Hughlings Jackson, of London, that we owe the physiological study of these cases, and of cases of more limited epilepsy, and the first demonstration of the dependence of localized spasms upon limited lesions of the opposite cerebral hemisphere.

Indeed, in prosecuting these clinical and post-mortem studies, Hughlings Jackson laid the foundation for the vigorous hypothesis of cerebral localization, as Ferrier states in the dedication of his book on the "Functions of the Brain," to this illustrious physician. So far as my own experience goes, autopsies have invariably verified the theory of localized epilepsy which I have stated, and the journals of the last five or six years contain numerous corroborative cases. As the evidence now stands, chronic localized convulsions must be looked upon as almost positive indications of a localized lesion in the opposite cerebral motor zone, most probably a tumor.

What I have said of localized convulsions applies to localized paralysis. It, like spasm, may be limited to a small muscular group, or to one half of the body; it may begin in a part and gradually extend. In general terms, paralytic phenomena follow in the wake of the convulsions at a distance of weeks or months, and have the same distribution.

Neuro-retinitis, or choked disk, is a frequent result of tumor within the cranium, but this symptom may, on the one hand, be absent with a large or even monstrous cerebral sarcoma, and on the other, it does not afford any indication of the locality of the tumor. The notion which was current a few years ago, that neuro-retinitis was pathognomonic of cerebral tumor, is wholly without foundation.

From my observations I am led to conclude that the occurrence of localized convulsions and paralysis, without choked disk, is valuable evidence of tumor, while choked disk without localized spasm and paralysis is merely a basis for suspecting tumor. The association of the two sets of symptoms makes up almost positive proof of the existence of a neoplasm. A diagnosis based on this symptom group is quite as secure as that of any other disease giving rise to local physical signs.

The value of headache, or localized cranial pain more strictly speaking, is also variable. By itself it is not strictly indicative of tumor, but with either the choked disks or with localized motor disturbance it becomes highly significant.

The co-existence of the three symptoms justifies a positive diagnosis of cerebral tumor.

Had I more time I should like to speak of the possibility of a still finer diagnosis in cases of tumor of motor districts of the brain. We are sometimes enabled, through recent advances in experimental physiology and pathological anatomy, to localize tumors within an inch or two of their actual situation, in the regions known as centres for speech, centres for the face, centres for the arm and hand, centres for the leg, and centres for both arm and leg. The future of neurological medicine is pregnant with discoveries in this direction, which will have very practical application.

My purpose in embracing the opportunity of addressing you was to make a sketch of the scientific and logical basis for progress in the direction of early diagnosis.

The affections whose semeiology we have studied—cerebral tumor, paralytic dementia, and posterior spinal sclerosis—are as yet incurable. Yet, if we can ever hope to apply remedies to them successfully, it will have to be done at the earliest moment when their recognition is possible by the general practitioner, who naturally has charge of the cases in their incipience.

REPORT ON THE USE OF HYOSCYAMIA AS AN HYPNOTIC AND DEPRESSO-MOTOR.*

I HAVE been instructed by the Committee on Neurotics to present a summary of the experience of its members with that powerful and seldom-used alkaloid—hyoscyamia. Due credit will be given at the proper place to the gentlemen who have contributed observations.

The report is divided into four parts. 1. A brief and, I must add, an incomplete sketch of the history of our knowledge of the drug, and a summary of the conclusions of the few physicians who have employed it. 2. A relation of our own cases testing the value of hyoscyamia as an hypnotic. 3. A relation of the cases showing the power of hyoscyamia as a depresso-motor or paralyzant. 4. Our provisional conclusions respecting its utility and the best modes of its administration.

I. HISTORICAL.

Hyoscyamia in an impure form was discovered by Peschier, in 1821,† and by Brandes about the same time.

In 1833, Geiger and Hasse ‡ found the pure solid alkaloid very much like the one we now use.

They determined its molecular constitution to be:

$$\text{Hyoscyamia} = C_{15}\ H_{23}\ N\ O_3,$$

and this formula is accepted as correct even by the latest authorities.§ By the same chemists it was shown that Hyoscyamia might be looked upon as a double body, in the same way as atropia, giving a formula:

$$\begin{array}{ll} \text{Hyoscin} & = C_6\ H_{13}\ N \\ \text{Hyoscinic acid} & = \underline{C_9\ H_{10} - O_3} \\ & \ C_{15}\ H_{23}\ N\ O_3. \end{array}$$

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* Read before the New York Therapeutical Society, February 13, 1880. Reprinted from the *Archives of Medicine*, vol. v., No. 2, April, 1881.

† *Annalen der Chemie und Pharmacie*, i., p. 333. ‡ *Idem*, vi., p. 270.

§ The *National Dispensatory*, second edition, 1880, p. 747.

Hyoscyamia is obtained from the leaves of hyoscyamus niger and hyoscyamia albus, in very small quantities; according to Schoonbroodt, about 0.164 per cent.; according to Wadgymar, 0.143 per cent., and according to Thorey (using the dried leaves picked before flowering), 0.188 and 0.208 per cent.* These experimenters and Merck, of Darmstadt, have greatly improved the processes for extracting the alkaloid.

In our market Hyoscyamia is found in two forms—both of Merck's manufacture:

First, a colored resinous extraction which is quoted at $2 a gram.

Second, a white crystallized, or semi-crystallized substance, very pure and exceedingly powerful, which costs, at wholesale, $11 a gram.

Both these forms of Hyoscyamia are dispensed by our leading pharmacists, and two firms supply reliable pills made of the crystallized form. Messrs. Caswell, Hazard & Co. offer it in the shape of tablets, each containing .0012, and Messrs. McKesson & Robbins make little pills, each containing .0012. Both of these preparations have been found reliable by your committee, the tablets of the first-named firm seeming to be a little stronger.

One of our number, Dr. Ball, has employed an amorphous preparation by Keith; a substance whose physiological properties are not well understood, and which may be a resinoid instead of an alkaloid.

The mode of administration will be stated in connection with the work of each observer.

I have consulted the following writings on Hyoscyamia:

The essay of Oulmont on the use of Hyoscyamia in paralysis agitans, tremors, etc., I have been unable to find, and have not even seen an abstract of it.

John Harley: "The Old Vegetable Neurotics," London, 1869, p. 321.

Dr. Harley's experiments with hyoscyamus and hyoscyamia are replete with interest. He used the tincture, succus, and solid extract of hyoscyamus, and made for himself a brownish, semi-crystallized sulphate of hyoscyamia. He experimented on mice, cats, dogs, and on man. The following are some of his conclusions:

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* Cited by A. and Theo. Husemann, "Die Pflanzenstoffe," pp. 478, 9.

1. Hyoscyamia differs somewhat from atropia, in not accelerating the pulse as much, and by a greater effect on the cerebrum and the motor centres.

2. Small doses will reduce the pulse-rate by 10 or even 30 beats. Larger doses cause a rise.

3. Sleep and great muscular relaxation are produced. To cause sleep, 45 cc. of the succus hyoscyami or 30 cc. of the tincture are required, and from .002 to .008 grain of hyoscyamia (hypodermically) will do the same.

4. In one case .50 of the solid extract produced a busy delirium not unlike that caused by hemp. The effects of Hyoscyamia are very like alcoholic intoxication.

5. Its action on the cerebrum is very similar to that of opium, except that it does not cause excitation of motor centres. No antagonism exists between the two.

6. Clinically, it is useful as a sedative to the heart. It is useless in convulsive affections. (In view of our own experience this last statement seems unaccountable.)

7. Hyoscyamia is excreted through the kidneys.

A. and Theo. Husemann, "Die Pflanzenstoffe," Berlin, 1881, p. 475. These authors give an excellent pharmaceutical account of the alkaloid, a rather meagre summary of the physiological researches of Schroff, Lemattre, Buchner, Fronmüller, Düllnberger, etc.

Their conclusion is that the action of hyoscyamia is qualitatively similar to that of atropia. Its chief use is as an hypnotic.

Schroff thought hyoscyamia produced more complete and permanent dilatation of the pupil than atropia.

Dr. Robert Lawson, Assistant Medical Officer to the West Riding Asylum, has contributed to the celebrated medical reports of that institution two elaborate papers on hyoscyamia; one, "On the physiological actions of hyoscyamine," appeared in the fifth volume, 1875; and the other, entitled "Hyoscyamine in the treatment of some diseases of the insane," in the sixth volume, 1876.

The following are some of the conclusions reached by Dr. Lawson in his first or physiological study. He used an amorphous hyoscyamia made by Messrs. Smith, of Edinburgh—evidently a weaker preparation than Merck's:

He found hyoscyamia to be more hypnotic and more diuretic than atropia.

Very rarely there occurred a rash on the face and forearms not unlike measles.

Small doses of hyoscyamia at first reduced the pulse rate, increasing the arterial tension, then excited the circulation. The axillary temperature fell 0.3° C.

Large doses caused immediate rise of pulse, delirium, motor paralysis, sleep, and produced diuresis.

Dr. Lawson's conclusions in his second or clinical study are:

Hyoscyamia is useful in cases of insanity with delusions of suspicion and in mischievous cases.

Remarkably good results are obtained in the status epilepticus.

In the excitement of general paralysis large doses, .015 and .03, are required and do good.

Small doses are efficacious in chorea and in locomotor ataxia.

Hyoscyamia is useless in acute melancholia, in acute mania with delusions of suspicion, and should be avoided in furious mania. As results of the long-continued use of small doses of hyoscyamia he observed impairment of appetite, but not much dryness of the throat. He employed Merck's amorphous alkaloid (extractive), given in the following formula:

℞.	Hyoscyamiæ,	.045,
	Spt. etheris,	.25,
	Spts. vini rect.,	1.2,
	Aquæ font., *ad.*	62.cc.—*ft. haustus.*

I might also mention that Dr. W. Bevan Lewis, assistant medical officer to the West Riding Asylum, in a paper upon "Calorimetric observations upon the influence of various alkaloids on the generation of animal heat," published in W. R. Asylum Medical Reports, Vol. VI., for 1876, makes the following statement respecting hyoscyamia (page 51):

Hyoscyamia in small doses diminishes heat-formation, while in large doses it greatly increases it.

Gubler, "Leçons de Thérapeutique," Paris, 1877, pp. 138, 9, refers very briefly to hyoscyamia. It causes less delirium, and more often a natural sleep than do atropia and daturia.

Refers to Oulmont's use of hyoscyamia for various tremors.

Denies that it is to be had in a pure state.

Nothnagel and Rossbach, "Handbuch der Arzneimittelehre," 1878, p. 666, state that its physiological action is similar to that of atropia, and refer to its use in tremors. They say nothing of its use as a narcotic. The dose is the same as that of atropia.

Wood, "A Treatise on Therapeutics," 1880, does not state doses for internal administration. He does not believe that any difference between the action of belladonna and hyoscyamia has been proven.

Bartholow, "Materia Medica and Therapeutics," 1880, gives a fair account of physiological actions of hyoscyamia, but is not sufficiently explicit in giving doses of the two kinds. Strangely enough he classes hyoscyamia among excito-motors.

Stillé and Maisch, "National Dispensatory," 1880, pp. 747-9, give a good account of hyoscyamia, clinical and physiological. Its action on the spinal cord is paralyzing, like that of conium. They refer to Lawson's use of it in mania. Dose, .001 repeated. In violent excitement, up to .06.

A more recent article is one by Dr. Geo. H. Savage, of Bethlehem Hospital, in the *Journal of Mental Science* for July, 1879. Dr. Savage does not like hyoscyamia in the treatment of psychoses, but apparently largely because of certain preconceived general views against neurotics. He admits the quieting and hypnotic influence of the crystallized and the amorphous hyoscyamia, and he gives preference to the amorphous. The former he gave in doses of .0025 or .004, and the latter in doses of .008 to .06. In many of his cases anorexia was produced; in a few, collapse. In melancholia harm is done by hyoscyamia. This alkaloid is only good to secure temporary quiet and sleep.

Lastly, Dr. Prideaux, assistant physician to the Friends' Retreat, York, in several numbers of the *Lancet* for September and October, 1879, has summed up his experience with this drug. He noted the usual differences between small and large doses, viz.: small doses reducing the pulse-rate and cerebral excitement, followed by motor paralysis and sleep; large doses cause an immediate acceleration of the pulse, reduce the temperature, and produce profound sleep. Death may be produced by paralysis of the respiratory centre. Dr. Prideaux used hyoscyamia in several forms of mental disorder, and in the status epilepticus, and reached the following conclusions:

1. In most cases of mania, or where there exists great excitement of any aggressive or destructive character, or rapidity of movement and speech, the use of the drug is the most effectual

and rapid means of exercising that form of restraint which has been known as "chemical restraint."

2. That in cases of acute mania it will produce sleep and quietude when all other drugs have failed, and is one of the most rapid and reliable narcotics which we possess.

3. That in the treatment of the epileptic status in epileptic mania, it diminishes the number, frequency and severity of the attacks, especially if its administration be extended over some time.

4. That in delusional insanity, especially in the mania of suspicion, and in other forms of mania where the delusions are varying and changeable, it has a decided action in producing such an altered condition of the cerebral status that a condition which has been called "physiological mania" results, and this so eclipses the former delusions and hallucinations that they are forgotten, and the mind becomes clear; while if the subjection to the influence of the drug be continued, it ultimately leads, under favorable circumstances, to a permanent condition of quiescence and restoration to a healthy frame of mind.

5. That in chronic dementia, associated with destructive tendencies, bad habits and sleeplessness, the condition of the patient much improves after a continued course of small doses of the drug.

Dr. Prideaux also recommends that hyoscyamia be used in senile mania, delirium and meningitis.

He used Merck's extractive hyoscyamia, and gave from .004 to .03 and even .06 in solutions by the stomach.

II. CLINICAL EXPERIENCE WITH HYOSCYAMIA AS AN HYPNOTIC.

Dr. J. C. Shaw, Medical Director of Kings County Lunatic Asylum at Flatbush, has made a somewhat extensive trial of hyoscyamia in cases of insanity. The following is his summarized report to your committee:

I have always used Merck's amorphous alkaloid.

So far I have used the medicine in about fifteen cases.

Acute and subacute mania is the condition in which I expected most benefit from its use. I have also used it in chronic mania with excitement and destructive habits, in the excitement of general paretics, in the maniacal excitement following epileptic seizures, etc. This drug is of great service in acute mania where the patients, from loss of sleep and continued motion, soon become exhausted, and then follows a disagreeable train of symptoms, such as dry tongue, sordes on teeth, and perhaps diarrhœa. If the patient refuses food and has to be fed with the tube, this makes the state of matters still worse. Hyoscyamia is a remedy which obviates this very largely; it is very certain in its action; I usually begin by giving .015 to .03 grain by the mouth.

The following cases are given in a condensed form as illustrations:

CASE I.—Female, aged 34. Admitted December 4, 1879, to the Kings County Insane Asylum. Married; had one child 9 years ago. The attack has lasted two months. She has delusions of persecution; attempts to injure herself and others; talks incessantly in a loud voice; is incoherent; will not converse or answer questions; her general health is poor; she looks thin and pale; pupils and articulation normal; tongue clean; does not sleep; talks and screams all night; ceased to menstruate two months ago, just as attack began. December 6th, she begins to menstruate; December 7th, she has become still more noisy and restless; breaks windows and furniture, and strikes herself up against other patients; throws herself on the floor or up against the wall, evidently with suicidal intent. Two days after, the 9th, she has to be fed; tongue and lips have become dry; she talks and moves about continually. To-day, for the first time, we give her .03 hyoscyamia at 12 M. In one-half hour she was quiet. At 3 P.M. she was sitting upon a bench apparently fast asleep; eyes closed and head resting on chest; the cheeks puffed out at each expiration; pupils dilated; she was profoundly under the influence of the drug. At 8 P.M. of the same night she was fed with the tube, but went to sleep again immediately after it. When she awoke in the morning she again became noisy; passes her urine in her clothing. December 10th, was fed with tube and .03 hyoscyamia given; at 12 she was quite quiet, and ate a hearty supper that evening.

Dec. 11th. .03 given this forenoon, and she slept the greater portion of the day. She ate her dinner, but would not eat supper. As she is feeble, tongue and lips dry, she is fed. At 10 P.M. she is quite noisy; she is given 1.2 chloral and extra night-watch placed over her. She throws herself out of bed, and tries to strike her head against the floor and wall.

Dec. 12th. She slept last night; to-day .03 hyoscyamia is given her; she slept nearly all day; she will eat some of her meals; tongue is now moist and clean; she spits at every one who goes near her, and will not answer any questions.

Dec. 13th. The .03 dose appears to have such a profound effect upon her that we give her .015, which appears to have almost as much effect as the .03 did. She slept almost all day; at night, gave her 1.2 chloral and a bottle of ale. She did not sleep; restless, and talked all night.

Dec. 14th. Is noisy and running about. I feed her with the tube myself, and give her .015 hyoscyamia; tongue is moist and clean; she was quiet after the medicine, but did not sleep.

Dec. 16th. She did not sleep last night and was restless; so this morning she is given .015 hyoscyamia, after which she slept.

Dec. 17th. Noisy and excited; gave .015 hyoscyamia.

Dec. 18th. Removes her clothing and is noisy; .015 given.

Dec. 19th. Eats much better; has not to be fed; talks incoherently; .03 hyoscyamia given at bed-time in a bottle of ale. Slept all night.

.015 hyoscyamia is given every day until Dec. 27th, producing its physiological effect each time.

On Dec. 27th none is given.

Dec. 28th. .015 given, and every day after until Dec. 31st.

She is much improved physically; eats her meals; is quiet.

The hypnotic effect of the drug is here well shown by this case, and its great superiority to chloral in that respect.

CASE II.—Male, aged 36. Admitted Nov. 8th. Certificates state that he declares that he is the Supreme Being; the Virgin Mary is his wife and mother. On admission is very noisy, shouting, clapping his hands, walks about incessantly, and behaves in a very demonstrative manner; is incoherent; talks about the priests, Virgin Mary, etc., in a rambling manner.

He was given .03 of hyoscyamia, and was quiet in fifteen minutes, and the full physiological effect of the drug is obtained; he sleeps profoundly for hours; as soon as he escapes from under the influence of the drug he is again noisy.

November 12th and 13th he is given .03 night and morning; he is noisy just as soon as the effect passes off.

It will suffice for me to say that the effect of the drug became more and more transient, and the dose was increased until, on December 2d, he took, at 8 A.M., .25 at one dose, the effect passing off so rapidly that at 3 P.M. he was again as noisy as ever; at 7 P.M. he is given .25 more, making .50 of hyoscyamia in one day. He no longer sleeps under the influence of the medicine as he did when it was first given; the medicine is discontinued as there is no permanent effect. During the time he was taking the large doses he passed large quantities of pale urine, which ceased as soon as the medicine was discontinued.

December 8th, he is given .06 hyoscyamia (there has been an interval of 8 days since he took the last dose, and he has been just as noisy and restless as he ever was, night and day) at 3 P.M., which in half an hour produced its marked physiological effects; but the effect of the subsequent doses becomes less and less marked, until on December 29th he takes .18 in the morning and .18 in the evening; .36 in the day, and as he has become so tolerant of it, it is again stopped.

CASE III.—A gentleman who had suffered from epilepsy for years past, has attacks of maniacal excitement after the fits, which will last 3, 4 days or a week ; nothing has ever arrested these maniacal attacks. He has a fit, and a few hours after he begins to shout psalms and praises to God. I gave him .03 hyoscyamia at 10.50 A.M ; at 11.20 he is unable to stand ; at 11.40 can sit up no longer, and has to lie down, and immediately sinks into a deep sleep, occasionally snoring; pulse 88 (112 before he took the medicine); increased reflex action, pupils slightly dilated. He sleeps until 6 P.M., when he eats his supper and goes to bed; he has to be assisted to his room, as he is unsteady on his feet; he sleeps all night soundly; the next morning is quiet, and in the afternoon is returned to the quiet hall. The attack would have lasted many days had we not given the hyoscyamia. This is one of the best results I have had with the drug.

I have tried it in some cases of melancholia with excitement, but without special benefit.

CASE IV.—Dementia following mania in a man of 50. He was in the asylum 20 years ago; was admitted this time about a year ago. He was in the habit of tearing up his clothing, bedding and bed; in fact, anything he could get hold of. If his hands were restrained he would use his teeth, kick the walls and break the furniture.

I gave him .06 hyoscyamia; in half an hour after he could not stand properly, walked about and tried to pick things off the floor, but which he was often unable to do; he staggered as if he was drunk. He was evidently conscious that there was something the matter with him, for when one of the attendants told him that he was drunk he laughed. In one hour he was completely unable to stand, but lay down on the floor and went fast asleep.

On the two following days we gave him .06 dose each day and then stopped. He never tore up any more things, and became one of the most quiet and orderly men in the hall, and would help to do some of the work.

I have used it in numbers of other cases, and the effects have been alike throughout.

The effects are temporary as a rule; most patients become quickly tolerant of it, and the dose has to be increased. There does not appear to me to be any deleterious effect produced by its use.

In acute maniacal attacks found inside and outside of asylums, it is undoubtedly a most valuable medicine, and certain in its action.

CASES REPORTED BY DR. A. B. BALL.

The two cases here reported illustrate the marked efficiency of hyoscyamia as an hypnotic in acute and subacute mania, the influence of the drug in controlling delusions, and its occasional tendency to paralyze the bladder.

CASE I.—The first case was one of acute senile mania in a gentleman sixty-five years of age, who had been an epileptic for years, but had had no convulsions for fourteen months. For several months albumen and casts had been occasionally detected in the urine, and granular kidney had been suspected. During the attack of mania, to be described, the urine was normal in quantity, free from albumen and casts, and no uræmic symptoms were present. On October 5, 1879, after having been restless and sleepless at night for three days, he suddenly became very violent, extremely voluble, and imagined he had been attacked and beaten by robbers. This delusion continued through the attack. Pulse and temperature normal for first three days, but both rose on the fourth day, and for the next fortnight the pulse ranged from 115 to 140, and temperature from 38.4° C. to 39.7° C. On October 11th he became comatose, and remained so for forty-eight hours, death being almost hourly apprehended. He recovered from this condition, however, and gradually gained strength, but the delusions continued as active as ever. Chloral, bromide of potassium, and morphine were used to procure sleep, but with very unsatisfactory results. On October 25th .01 of Keith's hyoscyamia was given at eight P.M. and repeated at nine. After the second dose, without any noticeable physiological effects, except the production of sleep, patient slept quietly for fourteen hours. Awoke more composed and rational. The same doses were repeated the following night, and on awaking after twelve hours' quiet sleep, the restoration of the mental faculties was complete. When closely questioned in regard to business matters, past events of his life, the multiplication table, etc., his replies were entirely satisfactory. He remembered the events of the first three and last three days of his illness, but the rest was a complete blank. No relapse occurred, and he has since remained in perfect mental health. During the entire illness the patient was attended by Dr. Alonzo Clark in connection with myself, and was also seen in consultation by Dr. E. C. Seguin.

CASE II.—The second case was one of melancholia of several months' duration in a gentleman about 70 years of age. Early in the morning of January 5, 1880, after having been unusually restless by day and sleepless at night for several days, he attempted suicide by jumping out of the second story window. Falling on the balcony of the story beneath, he escaped injury beyond a few trifling bruises. During the following day he was very restless and had delusions of persecution. At night .02 of Merck's amorphous hyoscyamia was given, and patient slept quietly for ten hours. On awaking he was much confused, staggered in his gait, and was unable to pass water. Catheter used. The symptoms of muscular paresis passed off in two hours. Patient more composed and rational. .01 given the next night. Slept six hours. Retention of urine again, but has general muscular paresis. Mental condition much improved. Increased appetite. During the next few days he became more tolerant of the hyoscyamia, and .03 doses were required to produce sleep, but as the medicine always produced retention of urine, it was stopped, and on Jan. 15th 1. of chloral with 2. of bromide of potassium were substituted. A more restless night. At eight o'clock A.M. he sat up in bed and ate his breakfast, after which he lay down in bed and fell into what was supposed to be a deep slumber. At nine o'clock he could not be roused, and a few moments later died. The cause of death was uncertain, as no *post-mortem* was allowed. Dr. Abram Du Bois attended the case, throughout the illness, in connection with myself.

A CASE OF DELIRIUM TREMENS, BY DR. F. P. KINNICUTT.

The attack occurred in a gentleman aged . . . The treatment was initiated on November 26, 1879, and consisted of large doses of chloral and bromide of potassium, with unsatisfactory result in producing sleep ; not more than two hours' sleep being obtained at any one time, and that of a restless character. During the night, November 1st and 2d, he took 5. of chloral and 8. of bromide of potassium without producing more than a few moments' restless sleep at any one time.

November 2d. The patient's condition is wretched. He is extremely restless, depressed, threatens suicide and even makes an attempt ; begs for stimulants. Hyoscyamia .0012 (Merck's crystallized preparation) is given by the mouth and ordered repeated *pro re nata.*

November 3d. Passed a very good night ; sleeping several hours very naturally. .0012 had been repeated in the afternoon of the 2d, and .0006 before 10 P.M. From this date there was continued and rapid improvement; good nights with apparently physiological sleep. .0012 administered by the mouth twice a day was found to be sufficient.

Aside from hypnotic action of the drug, the only effects observed were hallucinations of a pleasant kind, and slight dryness of the throat. No alteration of temperature occurred after doses.

CASE CONTRIBUTED BY DR. ANDREW H. SMITH.

I was consulted by letter on January 5, 1880, in regard to the following case :

E. T., aged 65. Three of his brothers and sisters died insane, two of them by suicide. For the past two years he has been gradually developing melancholia, recently becoming rapidly worse. He was very restless, sleepless, unmanageable, wandering about the house, talking wildly and excitedly. Pending removal to an asylum I advised the administration of .0012 of crystallized hyoscyamia to be given night and morning. This produced sleep at night and rendered the patient quiet and manageable during the day, to the great relief of the family, to say nothing of himself.

CASE OF MORBID DREAMS, BY DR. E. C. SEGUIN.

A gentleman, 50 years of age, affected with paræsthesiæ in the head and tinnitus aurium, was sent to me for treatment by Dr. J. S. Jewell, early in November last. Mr. X. was unquestionably hypochondriacal to a certain extent. Many years ago he had been a dyspeptic ; he had worked unremittingly at his business for many years, and in the last ten years his head had become worse and worse ; insomnia had developed and he had led a wretched existence. His insomnia was made the more trying because what little sleep he obtained was troubled by fantastic and fearful visions of a panoramic character ; scenes of violence, obscenity, comicality, would, as it were, pass before him almost with the vividness of hallucinations. Indeed, he had the same "visions" at any time if he dozed for a minute in his chair. Treatment began November 14, 1879. Injections of .18 of camphor failed.

On the 15th I gave him, chiefly with the idea of procuring sleep, the crystallized hyoscyamia in doses of .001, by hypodermic injections, at bedtime.

19th. Much better; little pressure in head for four days; less tinnitus; some sleep. At night, .002 of hyoscyamia hypodermically.

21st. Depressing and fearful dreams are much less marked, though there was not much sleep last night. Given .0024 hyoscyamia and .009 sulphate of morphia under the skin.

22d. Had a good night.

On the 29th he was ordered one tablet of .0012 hyoscyamia at bedtime with opium. The peculiar dreams have almost ceased. After this date, to the present time, Mr. X. has used one tablet of hyoscyamia almost every night, and has had ugly dreams only on one or two occasions. He has used small doses of McMunn's elixir of opium (morphia and opium in substance caused great itching) and a varied restorative treatment, with marked influence upon all his symptoms.

A parallelism might, I think, be drawn between this arrest of morbid dreams and the happy effect of hyoscyamia in cases of delirium tremens, delusions of persecution, etc.

HYOSCYAMIA AS A DEPRESSO-MOTOR.*

In the preceding paper I have presented a *résumé* of the state of our knowledge on the subject of the hypnotic action of hyoscyamia, together with the experience of the Committee on Neurotics of the New York Therapeutical Society. The following statements are with reference to the almost equally important depresso-motor or paralyzing effect of the drug.

It may be well to premise that in 1869, Dr. John Harley wrote: "In convulsive affections, it (hyoscyamus) has proved useless in my practice. The plant undoubtedly exercises a considerable depressing influence on the corpora striata, but it fails to diminish the excitability of the spinal centres, if it does not actually exalt it."† Harley, however, speaks of hyoscyamia as valuable in cardiac functional disorders, in neuralgia, especially visceral, in cardiac and pulmonary asthma. Oulmont‡ was more fortunate in his clinical experience. He employed Merck's crystallized hyoscyamia, in doses from .001 to .003 several times a day, in pillular form or hypodermically, and gave enough to obtain full physiological effects. He treated several cases of neuralgia, occipito-cervical, sciatic, etc., and nearly all the cases were cured pretty rapidly. In mercurial tremor, even in cases which had resisted sulphur baths and iodide of potassium, he obtained four cures and two reliefs. A case of progressive locomotor ataxia was not favorably influenced by hypodermic injections of hyoscyamia, excepting that the pains were somewhat dulled. A severe and rapidly fatal case of traumatic tetanus received doses which Oulmont considers to have been too small; the pain of the spasms was moderate, but the tetanus remained unchanged (this is in accord with Harley's statements). He had no opportunity of treating paralysis agitans, though, strange to say, he is often referred to as having been the first to use hyoscyamia in that disease; he merely refers to Charcot's experience.

This trial of hyoscyamia by Charcot is more explicitly stated by Ordenstein in his essay on paralysis agitans and sclerosis.* He says: "We have yet to mention a last therapeutic experiment. M. Charcot has recently given [to cases of paralysis agitans] two or three granules of about .001 of hyoscyamia per diem. This medicine has procured several hours of rest to a number of patients. New observations are requisite to enable us to express an opinion as to the value of this treatment."

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* Reprinted from the *Archives of Medicine*, vol. v., No. 3, June, 1881.

† "The old Vegetable Neurotics," London, 1869, p. 340.

‡ *Bulletin de Thérapeutique*, tome lxxxiii., p. 481, 1872.

Dr. Lawson† thought that hyoscyamia had proved serviceable in chorea and locomotor ataxia.

My own experience has been as follows:

HYOSCYAMIA IN PARALYSIS AGITANS.

CASE.—Mr. F., aged 42 years, consulted me, at Dr. Blumenthal's request, on May 19, 1879, and gave the following history: Good health until 1874, when he had a severe attack of "inflammation of the bowels" lasting for several weeks. A fistula in ano formed not long afterward and has continued. Without any other apparent cause he began to tremble in 1875, the fingers of the left hand being first affected. Not long afterward the left leg became affected, and in the course of two years, very gradually, the right hand and arm became involved. The symptom has steadily increased in intensity in both

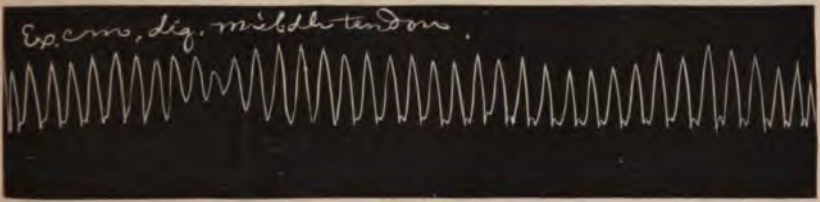

FIG. 1.

Tracing of movements of middle tendon of the extensor communis digitorum, in paralysis agitans.

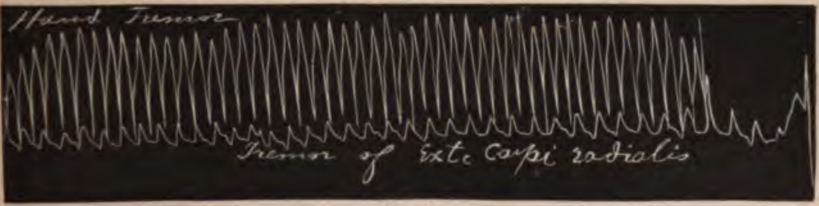

FIG. 2.

Upper tracing shows regular flexion and extension movements of the hand; the lower, pulse-like tracing represents the tremor of the extensor carpi radialis longior taken on its tendon; paralysis agitans.

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* Ordenstein, L. Sur la paralysie agitante et la sclérose en plaques généralisée, p. 31. Paris, 1868.

† West Riding Lunatic Asylum Reports, vol. v., 1875.

upper extremities; the left leg is about as it was three years ago, and the right lower extremity is unaffected. The legs feel weak, but patient has noticed neither pro- nor retro-pulsion. No numbness of limbs, headache, dizziness, loss of memory, or affection of general health. Parents did not tremble.

Examination.—General condition excellent. Facies is injected and exhibits the characteristic stare of the disease. No anæsthesia of face or hands. Dynamometer test of strength of grasp made useless by injury to right hand; no apparent loss of power. The tremor affects the left leg (slightly) and the two upper extremities; the head and right leg are perfectly still. The movement is constant, consisting of alternate movements of flexion and extension (mostly) made with great rapidity and with absolute rhythm. The rhythmic nature of the trembling is well shown in the annexed graphic tracings. Emotion makes the tremor worse; it can be temporarily checked by the will, and co-ordination of movements is exact, *i.e.*, there is no ataxia; he can place a finger on his nose with closed eyes, and can carry a full glass of water to his lips. The hands when resting naturally on the knees make the well-known pawing movements. The normal use of the hand for eating, dressing, etc., is much hampered by the tremor and by a certain slowness and stiffness in willed muscular movements. The patellar tendon-reflex is normal, or perhaps subnormal.

It may not be uninteresting to add that Mr. F. has been under the care of several eminent specialists for diseases of the nervous system, two of whom had looked upon the disease as sclerosis of the brain and cord. (This is a good illustration of the foggy notions which prevail as to the meaning of the forms of tremor and the diagnosis of disseminated sclerosis.) He had attempted a great variety of treatment, perseveringly, without relief, by means of, for example, sedatives, counter-irritants to the spine, hypodermic injections of strychnia, etc.

I told the patient frankly that nothing would cure him, but proposed a trial of hyoscyamia as a means of relief. The drug was administered for several weeks, hypodermically, and the following are notes of effects observed.

On May 21, 1879, I prescribed:

℞	Hyoscyamiæ, (Merck's crystallized)	.06
	Glycerinæ, Aquæ destillatæ, āā	6.00
	Acid. carbol. pur.,	.03

Mix, filter with care, and label:

"Hyoscyamia solution for hypodermic use, .06 = .0003."

This prescription was dispensed by Messrs. Caswell & Hazard, and in the afternoon of the same day I gave the patient an injection of .0012 in the arm. In the course of half an hour *all tremor had ceased*, the mouth was parched, and the pupils somewhat dilated. In an hour after the injection, the patient was greatly distressed by dilatation of the pupils and dimness of vision, by extreme dryness of the mouth, causing almost complete aphonia; and there was slight delirium. Four hours after the injection, these symptoms had in a great measure passed away, *but the hands were still absolutely quiet*, and the tremor only very gradually reappeared during the ensuing two hours.

Thus .0012 of hyoscyamia produced a complete cessation of the tremor for a period of at least four hours ; a result which was as surprising as it was gratifying to me, whose experience had been that no means known to our art, short of complete anæsthesia, were capable of arresting this trembling.

On subsequent days I injected from .001–.0012, with invariably the same results. In a week or two a certain degree of tolerance was established, and .0012 did not produce much distress, but still suspended the movements of the paralysis agitans for two or four hours.

The patient, and his relatives also, noticed that going up and down stairs, using hands for buttoning his clothing, feeding himself, etc., became much easier, and he felt stronger.

While the daily hypodermic injection of hyoscyamia, once a day, thus caused very great temporary relief, and produced a certain amount of positive continuous improvement, there were no unpleasant effects of any importance. The digestive organs remained in good condition, the accommodation was not wholly paralyzed, no abscess or special irritation was produced by the injections.

Early in July, pills containing .002 of hyoscyamia were substituted for the injections, with equally happy effect ; two pills a day giving the patient *several hours of absolute freedom from tremor*, without unduly severe toxic symptoms. During the month of August the patient became more sensitive to the drug, and the doses were reduced to .002 in the morning, and .001 in the afternoon. The paresis of accommodation varied somewhat from time to time, but was easily corrected by weak convex glasses. Prof. H. Knapp was consulted upon the question whether permanent injury to the eye might result from the long-continued or indefinite use of the medicine, and he replied that it could not.

The patient returned to his home in Louisiana, in September, taking with him a number of pills made at my request by Messrs. McKesson & Robbins, each containing .0012 of hyoscyamia.

I have since indirectly heard that the disease has made progress.

I have used hyoscyamia in several other cases of paralysis agitans, and always with the same effect, viz., temporary relief. The drug has been given in these other cases by the mouth, using the tablets of .0012, made by Messrs. Caswell & Hazard. One tablet in the forenoon and one at bed-time have been sufficient to almost arrest the trembling for a period of time varying from one to three hours, and to cause a delusive sense of improvement throughout the period of treatment. After a few weeks, on omitting the drug, it was observed that the tremor was as before. Indeed, I should add that in all my cases the usual progressive development of the disease has not been prevented.

Still it is a real addition to our therapeutics to have a drug which can, for a few hours, afford physical rest to a patient tormented by paralysis agitans. The hypodermic injection of [illegible] to .001 will surely do this; and the internal administration of from .001 to .002 will produce a similar less-prolonged suspension of tremor.

HYOSCYAMIA IN CHOREIFORM AFFECTIONS.

CASE I.—*Co-ordinated rhythmical hysterical spasm (hammering or pounding chorea).*

Mrs. K., aged about 29 years, was placed under my care during the later part of June, 1879. I learned that she had been hysterical for many months; having weeping, globus hystericus, at times pseudo-coma with rigidity, and nearly constantly of late a peculiar spasmodic affection which will be described in detail. This spasmodic movement was at the time I saw the patient almost the only symptom present. Appearing late in the winter, it had changed its character and rhythm a good deal, but had never been completely suspended. Mrs. K. had suffered from no corporeal disease, except what, from the imperfect account I received of it, may have been imperforate hymen, which had been successfully operated during the spring by a prominent gynecologist.

When I first saw Mrs. K., she was sitting up in bed, looking pale, but not much emaciated; her eyes were bright, and she was perfectly clear-minded and reasonably calm. A pillow lay upon her knees, and she was pounding it with her two closed fists, with a regular up-and-down stroke. The blows were quite hard, given with perfect rhythm, both hands coming down together, at the rate of one hundred strokes per minute on the average. There was no marked motion above the elbows; it was a regular hammering or pounding. The excitement of the examination momentarily increased the rate of hammering, and this also occurred on one side when the other hand was forcibly held by the observer or kept still by volition. The will could for a few moments suspend the movements, but the effort caused distress, and was followed by increased rapidity and force of striking. The noise made by the pounding could be heard a long distance, and on several occasions, when the pillow had slipped out of position, Mrs. K. had bruised her knuckles.

I was informed that this extraordinary performance was kept up all day and evening, until sleep supervened. The trained nurse in attendance estimated that the pounding was done for fourteen hours on some days, and eighteen hours on others. A simple calculation will show that from eight to ten thousand double blows were struck each day by this lady. There was no apparent fatigue from this wonderful expenditure of motor force; and several times visiting the patient late in the evening I found her nearly as fresh-looking and as amiable as in the morning. She was tastefully dressed nearly the whole day, but sat on a lounge and occasionally took a few steps around the room, hammering all the time.

Occasionally, almost each day, the hammering ceased, and was replaced by tetanic rigidity of the extremities and trunk, with or without seeming coma; sometimes with emotional excitement.

I made a thorough examination of the patient, except, owing to the patient's refusal, of the sexual organs, with a negative result. The urine was especially examined and found free from signs of renal disease.

The case, after making marked progress in relation to the hysteria and the special spasms, terminated in a most tragic manner during my absence from New York one Sunday, by uræmic convulsions and coma. A *post-mortem* study of the urine showed albumen, hyaline and granular casts.

I desire to report the effect of hyoscyamia upon this choreiform affection, premising that no medicine tried by the several physicians who had treated the case before me had arrested the hammering.

The solution employed was that for which a formula is given. On June 28th, at 1 P.M., I injected .0006 of the solution under the skin. At 1.40 there was marked flushing of the face, dilatation of the pupils, dryness of the mouth; the pulse unaffected, beating sixty-five per minute. *The hammering was reduced to the slightest blows*, given with great regularity. This reduction of the spasm occurred very gradually, from very hard pounding at the rate of more than one hundred blows per minute. The patient complained of a sense of weakness in the arms, and of drowsiness, and fell asleep for three hours. The pounding did not reappear until some time after waking, and then it was weak. At 9 o'clock P.M. I made a second visit, and found the patient comfortable—pounding very gently. I then injected about .0005, with the same gratifying results, viz., moderate toxic effects, and complete cessation of spasm with good sleep.

These effects were regularly obtained each day for a fortnight, but at the end of that time it became evident that the effect of hyoscyamia was only a palliative, as in paralysis agitans.

CASE II.—Chronic chorea in the adult. Male, aged 45 years; employed as a helper at a railway depot for the past twelve or fourteen years; has been the subject of most marked general chorea. During these years he has had choreic movements affecting the extremities, neck, face, tongue, jaws, etc.,—in brief, the whole body. In the last few years sleep has been greatly interfered with by the continuance of movements at night. Even when asleep, he has jerked about in bed to such an extent, striking out with his arms and legs and grating his teeth, that his wife had been unable to occupy the same bed.

Examination showed only common chorea. No paralysis, anæsthesia, or other signs of organic disease of the nervous system. Speech affected as in common chorea, not as in sclerosis. Facies and manner somewhat demented. Intelligence fairly preserved. Examination is made difficult by extreme choreic disorder.

January 7, 1880, he received .0006 of hyoscyamia, hypodermically, at 9 P.M. The only effects were considerable dryness of the mouth and some incoherence in the night.

January 8th. At 10.10 A.M., pulse, 74; axillary temperature, 36.5° C.; respiration 20; pupils, 4 mm. diameter. Can hardly keep feet and hands still an instant. The mouth and tongue are in such constant motion that it is impossible to take temperature in the mouth. Administered .0008 of hyoscyamia.

At 11.10 A.M is sound asleep and perfectly quiet; a state of things not seen in the last eight years. Pulse, 82; pupils contracted. After being awakened he remained fairly quiet, and would protrude the tongue, a thing impossible before. Pulse then, 90; skin, warm; and axillary temp., 36.7° C. Pupils, wider. From this time on, the administration of .0015 morning and evening kept the chorea almost completely in abeyance, and procured good sleep. The drug began to affect him in about half an hour, and in two hours a maximum effect was reached.

Later, for a day or two, the dose was increased to .002 and even .003. The chorea was completely suspended by these doses, but excessive dryness of the mouth and nausea were produced.

Once the treatment was suspended for forty-eight hours. The chorea returned to a moderate extent. Later, the use of hyoscyamia was wholly suspended, partly because of nausea and also because of the addition of Fowler's solution to the treatment. The arsenic was given hypodermically twice a day, in doses of .60 finally. Some toxic effects were manifested. About March 1st the patient returned to his home in the country decidedly improved, and was able to resume work. He was seen in the middle of March, 1881, and proved to be very much less choreic than when treatment was instituted.

During the course of the treatment by means of hyoscyamia a number of observations on the pulse, respiration, and temperature were made, with the same result as on the first two days. Two experiments were also made with the view of determining whether the patellar tendon-reflex was affected by the drug.' On February 13th at 9 A. M. the reflex was found normal. Administered .002 hyoscyamia under the skin. In half an hour there was great dryness of the mouth, dizziness, staggering, and sleepiness. Went to bed and slept soundly for two hours without a movement. Awoke, talked in a flighty manner, and went to sleep again. Was seen at 2 P.M., still moderately under the influence of the drug ; tendon-reflex at knees, normal. At 5 o'clock P.M., reflex normal. Gave .002 again, with full physiological effects. In this condition, extreme dryness of mouth, dizziness, staggering gait, hallucination of hearing (heard a cat), great congestion of face and neck, violent headache, and dilated pupils ; knee reflex remained normal.

I am indebted to Dr. R. W. Amidon for the details of this observation.

In many of the reported cases of mania treated by means of hyoscyamia, whether given by the mouth or hypodermically, muscular relaxation is mentioned as a prominent symptom following a full dose. (*Vide* cases by Dr. Shaw, p. 477.)

These cases demonstrate, it seems to me, that hyoscyamia is a powerful and constant depresso-motor or anti-spasmodic. The spasmodic movements of paralysis agitans, of hysteria, of chorea, can be completely arrested by it for several hours. The power of retaining the erect or sitting posture may also be annulled by hyoscyamia. These facts and Dr. Amidon's observations on the state of the tendon-reflex during the toxic state produced by hyoscyamia would seem to justify the conclusion of Dr. John Harley, cited at the opening of this paper, that the "plant undoubtedly exercises a considerable depressing influence on the corpora striata, but it fails to diminish the excitability of the spinal centres, if it does not actually exalt it."

The experience of all observers, with reference to the physiological and therapeutic effects of hyoscyamia, may be summed up as follows:

1. It acts as a mydriatic, but whether more fully or longer than atropia remains to be settled.

2. When given in small doses it reduces the cardiac pulsations, increases arterial tension, and checks the loss of body heat. It also produces hallucinations and delirium. It may cause a fall of axillary temperature, and occasionally a rash.

3. In large doses it immediately increases the pulse rate, produces a seeming paralysis or motor debility, and sleep.

4. Hyoscyamia is indicated in mania, restlessness, delusions of persecution, dementia with agitation and destructiveness, epileptic mania, insomnia, rapid action of the heart, epilepsy (?), status epilepticus, chorea, paralysis agitans, hysterical spasms, tremor, neuralgia, etc.

5. In mania and allied states it produces sleep as certainly, or even more certainly, than chloral, without any evil after-effect, unless it be occasional gastric disorder.

6. In cases of delusions of persecution or of suspicion it has produced a positive cure.

7. In paralysis agitans it achieves what no other remedy ever has done, viz., it arrests the movements for four hours or more without producing insensibility.

8. In the status epilepticus it shortens the attack materially; perhaps better than any other single remedy.

9. It is a diuretic of no mean power.

10. The curative power of hyoscyamia does not appear to be great. In some cases of insanity its use has been followed by recovery; but, as a rule, we must look upon it as a good narcotic, often speedier, more complete, and less objectionable than morphia and chloral hydrate.

11. In spasmodic diseases, so far, we can speak of hyoscyamia only as an ameliorating agent, or as a palliative.

I would suggest, lastly, that in cases of very acute chorea, where death is threatened by incessant motion, hyoscyamia, given hypodermically, may prove of benefit by securing muscular relaxation with certainty, thus allowing the patient to rest, and giving time for other remedies to act.

THE PHYSIOLOGICAL EFFECT OF ACONITIA IN POSTERIOR SPINAL SCLEROSIS: CAN IT BECOME AN AID IN DIFFERENTIAL DIAGNOSIS?*

I HAVE observed in six well-marked cases of posterior spinal sclerosis, in the first and second stages, a remarkable resistance to the action of aconitia as shown by numbness of the periphery.

These six patients took large doses of the alkaloid, from three to six tablets of .0006 each, in a day, without numbness in the ataxic or neuralgic parts. Numbness showed itself in the parts of the body above the supposed seat of sclerosis, and several of the patients felt faint, dizzy, and quite sick from the medicine.

Dr. W. R. Birdsall, at my request, administered aconitia in full doses to several ataxic patients under his charge with substantially the same effect; one case experienced no tingling; another case had a little numbness in toes; and a third case, after taking four doses of .0006, used at intervals of three hours, felt some numbness in ends of fingers; a few hours later was "numb all over."

It appears from these nine cases that tabetic patients are peculiarly insusceptible to the characteristic sensory disturbances produced by aconitia. This resistance, apparently absolute in some cases, is shown in the first stage of the disease. One of the cases, which took at one time .01 of aconitia in less than forty-eight hours, was examined *post mortem*, and the cord found sclerosed. The aconitia used in these tests was Duquesnel's crystallized aconitia, prepared by Caswell, Hazard & Co., in tablet form. The specific effects of these tablets were obtained during the same period in other cases of disease and in healthy patients. For example, in my own case, .0006 at 10 A.M. and at 12 noon, made me numb from head to foot, and chilly for nearly five hours.

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* Read before the American Neurological Association, June 17, 1881. Reprinted from the *Journal of Nervous and Mental Disease*, July, 1881.

While not now prepared to advance a theory of the manner in which sclerosis of the posterior columns prevents the sensation of tingling and numbness in tabetic patients charged with aconitia, I feel confidence in my facts, and would offer them as constituting a new negative test or symptom of the disease.

Either there is an unknown lesion in the gray matter in ataxia, and in that gray matter the passage of the abnormal sensation is interfered with; or, second, that the drug does not act upon the gray matter, but upon the nerve fibres, and as these are diseased, the sensations do not arise.

A CASE OF DIPHTHERITIC ATAXIA AND PARALYSIS FROM ANAL DIPHTHERIA—CURE.*

Mr. B., aged 58 years, has enjoyed good health with exception of hemorrhoids. Never any fulgurating pains, or diplopia.

Nov. 12, 1880, was operated on for large hemorrhoids by injection of carbolic acid and oil. Reaction followed, with diphtheritic exudation on hemorrhoidal masses, chill, febrile movement, and much prostration. The anus was well about Thanksgiving (27th).

Early in December seemed fairly well, but a few days before Christmas legs were weak and feet numbish. Gradual increase in weakness of legs, and a few days before examination, hands became weak, awkward, and numbish. Bladder unaffected; no spinal or peripheral pain, or cincture feeling.

Examined January 25, 1881. Presents paresis of upper and lower extremities, with numbness and slight but distinct anæsthesia of feet, legs, and hands. The striking symptom, however, is the ataxia, which is typical both in hands and legs; no trace of patellar tendon-reflex. Pupils normal. During the ensuing two weeks the paresis increased, and gradually obscured the ataxia.

Feb. 5th. Lies quite helpless on couch, almost no voluntary power in arms or legs; sensory symptoms as above. No atrophy or degeneration reaction. Improvement in voluntary power began February 15th, and progressed steadily, with corresponding diminution of the anæsthesia.

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* Read before the American Neurological Association, June 17th, 1881. Reprinted from the *Journal of Nervous and Mental Disease*, July, 1881.

March 29th. Walks with a cane.

May 3d. Is practically cured; only remains of attack is a slight occasional numbness in soles of feet; no tendon-reflex.

May 17th. A trace of patellar tendon-reflex on both sides.

The treatment consisted at first in the use of belladonna and ergot; later nux vomica and iron. At the last a simple solution of strychnia in nitro-muriatic acid was given.

A thorough electrical treatment and massage were also had. Until March 16th galvanism was used only; stabile ascending current to limbs and spine. After this date faradism was carefully used on the recovering muscles. The massage was made proportionate to the paralysis, and in the last few weeks was vigorously done.

I looked upon the case as one of myelitis, probably infectious, with deposits of minute organisms around all the anterior and posterior nerve roots entering the spinal cord, probably first in the posterior segments of the cord, and the anterior afterward, judging from the succession of events. I was much interested in the case because of the difficulty of diagnosis. I was strengthened in my suspicion of diphtheria from the absence of pupillary symptoms and fulgurating pains.

A SECOND CONTRIBUTION TO THE STUDY OF LOCALIZED CEREBRAL LESIONS.*

In 1877 I reported to the American Neurological Association † a number of cases with accurate *post-mortem* examinations, illustrating the doctrine of localization of functions in the brain. Since that time I have made several similar observations, some of which have been published as isolated cases. In the past year two remarkable cases of cerebral tumor bearing upon the Ferrier hypothesis have been added to my records, and I think that the time has come to offer a second installment of facts in this department of medicine to the medical public. I shall first relate my last unpublished cases, and point out their significance, then reproduce in brief the isolated observations, positive and negative, which I have separately published.

I would only claim, in offering this second paper, to be adding a few trustworthy data to a mass of observations which tend to support the theory of cerebral localization. This theory or hypothesis can be established as true only by great numbers of pathological facts corroborating the results of experimental physiology and of anatomy.

Case I.—Mrs. I. D., aged 58 years, seen October 3d, 1880.

A strong, intellectual woman, who has enjoyed good health. In early spring was overworked and anxious about the outfit of a daughter who was to be married.

In May began to have a peculiar general headache (different from any she had had before), most marked in the occipital region, and always worst at night. She often complained of a sore, stiff feeling in the neck on rising in the morning. At times, in connection with headache, has had nausea and vomiting. This headache has been a prominent symptom ever since, amounting at times to agony.

Later in the month of May, or in the early part of June, there was noticed a trembling of the left hand; this increased, and was accompanied by evident loss of power. Relatives of the patient describe two sorts of movements of the left arm: first, a slight and nearly constant fine tremor; and, second, attacks of considerable jerking, so that the patient was obliged to hold the affected left hand with the right. Each day there were several such attacks, some lasting an hour.

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* Read by title at the seventh annual meeting of the American Neurological Association, June 17, 1881. Reprinted from the *Journal of Nervous and Mental Disease*, vol. viii., No. 3, July, 1881.

† P. 202.

Has grown steadily worse; more headache, marked paresis of the left arm, with some contracture, slight weakness of the left leg. Sight not so good as formerly, but there has been no diplopia, hemiopia, etc.

Last night the pain was intense through the mastoid regions, and in the whole of the head. Was given .01 sulphate of morphia occasionally, and by 10 A.M. to-day had taken .05; is semi-comatose, but still groaning from pain; the left hand and arm are semi-flexed and stiff.

Examination at 5 P.M. Patient is profoundly asleep, yet can be roused; respiration is slow and very irregular, but not of the Cheyne-Stokes type. When spoken to loudly, points (with right hand) to the sides of the head as the seat of chief pain; is able to swallow. The pupils are small and fixed, the right larger. The right internal rectus is weak. The left lower face is paretic. The left arm and hand are strongly adducted and semi-flexed on the thorax, and passive extension is difficult and painful. Legs extended, not stiff; both show good reflexes at the knees. Left hand and leg are less sensitive than the right. The pulse beats about 72 per minute, and is weak; the axillary temperature is 37.4° C. After the use of atropia, I was able to observe typical neuro-retinitis (choked disk) in both eyes; no hemorrhages. Urine contains a trace of albumen.

My diagnosis was tumor in the right cerebral hemisphere, complicated by morphia narcosis. I considered that very probably the tumor was in the median region of the hemisphere, in the so-called centres for the arm and leg, according to Ferrier's experiments and to recent *post-mortem* facts.

A great many notes were made during the progress of the case, but they only show the extraordinary variations in the state of the patient, which I and others have observed in cases of cerebral tumor. Some days Mrs. D. would be sitting up and very bright, and the next day might appear moribund.

On October 5th is up on a lounge, is bright and cheerful, though mind wanders at times ; headache has returned about the vertex. Can converge eyes well. Exhibits common left hemiparesis, with contracture, most marked in arm and hand. Ordered solid food and iodide of potassium.

October 9th. The left arm is completely relaxed and the tongue is straight.

October 10th. Growing steadily worse. Attacks of pain in the head, at times very severe, controlled by morphia and chloral. The arm is now completely paralyzed, with painful contracture of elbow and shoulder. No voluntary motion in left arm for forty-eight hours ; the left leg, which four days ago could be drawn up fairly well, is now nearly motionless. Left face is paretic, but tongue points straight. Answers questions, but wanders: wants to be dressed, to go out, etc. Wets the bed. Optic nerves choked as before.

October 13th. State of paralyzed limbs has varied from partial to complete paralysis. Extreme sensibility to narcotics.

October 15th. Sulphate of quinia produced delirium the other evening, and she is easily plunged into dangerous narcosis by morphia. Morphia .002+ and chloral .15 have *some* effect.

November 1st. Divergent strabismus and slight drooping of right upper lid. Speech very indistinct. Left hemiplegia as above. Delirious and semi-comatose at different times. Incontinence of urine and fæces.

November 4th. Greater coma and first appearance of fever. 7.30 A.M. Pulse, 162; respiration, 52. At 4.30 P.M., pulse, 136; axillary temperature, 39.2° C.; breathing moribund, *i. e.*, inspiration and expiration equal. Left arm in semi-flexion on chest, elbow and wrist limber, fingers decidedly contractured. At 10 P.M., respiration, 56; pulse, 160; axillary temperature (six minutes), 39.8° C. Right eye is in slight external strabismus and motionless; the left is in continual lateral motion; pupils medium sized, equal.

November 5th, 1 A.M. Respiration, 56; pulse, 176; axillary temperature, 40.15° C.; jaws firmly closed. Death occurred before daylight, and the temperature finally rose to 40.6° C.

No *post-mortem* measurements could be made.

The autopsy was made about ten hours after death by Dr. R. W. Amidon under my direction. Drs. W. R. Birdsall and C. Adam were also present.

Very little blood escaped on removing the calvarium. The

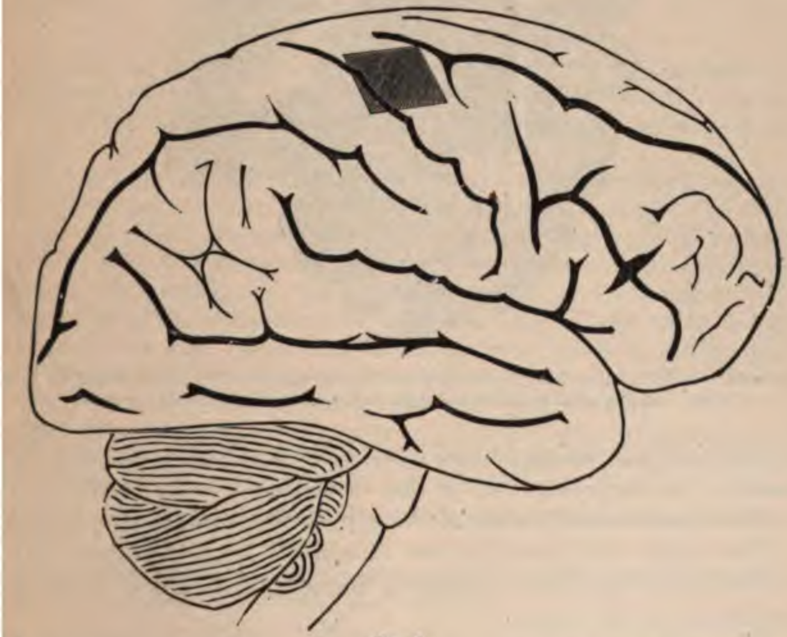

FIG. 1.

Lateral view of the right cerebral hemisphere, after Ecker. Shaded spot represents the location of the tumor. Superficially it involved only the ascending frontal gyrus.

pia mater was found excessively dry and sticky, and without gloss. There was a marked prominence of the right parietal portion of the brain, causing the whole hemisphere to appear much larger than the left. The convolutions about the upper end of the fissure of Rolando on the right side were very much flattened.

A vertical, transverse section passing through the middle of the motor zone revealed a consistent, grayish-red tumor, lying chiefly in the right ascending frontal convolution, wholly under the pia, and in the angle formed by the ascending frontal convolution and the paracentral lobule at the top of the brain. See Fig. 1.

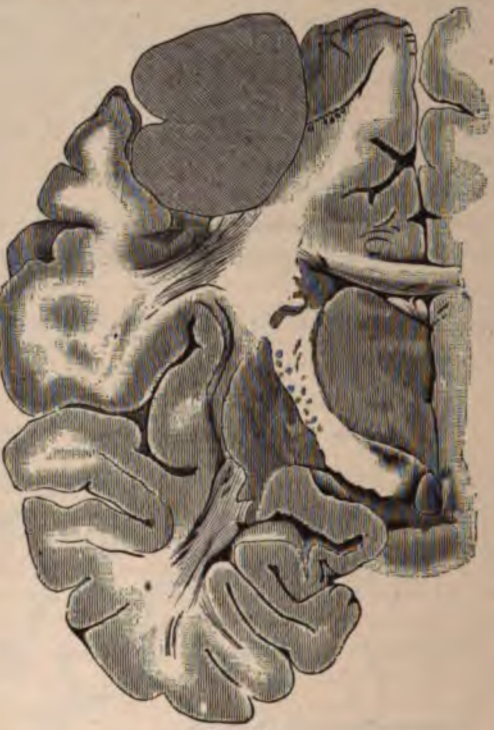

FIG. 2.

Transverse vertical section through the right hemisphere, anterior view; after photo. No. 5 of Bitot. The gray shaded mass in the upper part of the figure represents the tumor.

The tumor was about the size of a small English walnut, well defined from the brain substance, vascular, and at points almost gelatinous in structure.

The right third nerve was grayish. Right eye removed, showed an elevated papilla.

The brain and eye were placed in bichromate of potassium solution for hardening.

The following is a study of the topography of the lesion made upon the hardened specimens:

The tumor, ovoid in shape, lies in the upper part of the ascending frontal convolution and in its subjacent white matter. It measures upon the vertical transverse section of the brain, transversely, 15 mm. at its pia mater attachment, 20 mm. in its middle, and vertically, from its deepest point to the pia, 28 mm. See Fig. 2.

It extended well across the bottom of the fissure of Rolando, so as to slightly impinge upon the ascending parietal gyrus. The distance from the surface of the brain in the longitudinal fissure to the internal edge of the tumor is 25 mm., thus leaving the paracentral lobule and its attached white matter intact.

The tumor is spongy in texture, well defined from the surrounding cerebral substance, and seems firmly united to the pia. The microscope shows it to be an alveolar carcinoma.

It probably caused a great deal of pressure, in spite of its small size.

CASE II.—L. K., an upholsterer, aged 34 years, came to the Manhattan Eye and Ear Hospital, department for Nervous Diseases, October 6, 1879. He was a strong and healthy-looking German. The following is a transcript of my notes:

Has had attacks of right-sided epilepsy. First seizure was about two years ago (1877), and the attacks have occurred at the rate of one every four or six weeks. In the last few months has had attacks every week, and even several times a week. The phenomena have always been the same in these numerous attacks; the spasms being wholly restricted to the right arm and leg; the slightest attacks are only momentary shocks on the right side of the body—no spasm in the face. Even in the severe attacks the spasm is wholly clonic, and he never loses consciousness. An exception to this occurred on August 5, 1879, when he had a severe seizure with loss of consciousness.

The attacks last from a few seconds to a few minutes; they are preceded by a sensation of something rising from below upward to the throat, and there causing choking. He never froths at the mouth, or bites tongue, or micturates in attacks, and during them he is often able to speak a few words in a jerky manner.

In intervals between attacks has good use of his right hand and leg; he is now working at his trade. Mind clear and calm.

Very lately has noticed a slight weakness in the right limbs, and the right leg has been the seat of an indefinite numbness. Complains of diffused headache, mostly frontal. No vertigo or petit-mal.

Denies injury to head and any venereal disease.

Examination.—Manner, appearance and speech normal. No facial palsy; tongue straight; pupils equal. Right hand grasps 45° and 48°, and the left 45° and 45° on Mathieu's dynamometer. No anæsthesia to careful testings. Patellar tendon-reflex absent on the left side, and strong on the right (never sharp pains in legs). The walk is rather of hemiplegic type on the right side; the right foot is held slightly in equino-varus position. Complains of sight of right eye, and states that when a soldier he was obliged to aim with the left eye. Examination of eyes by Dr. J. O. Tansley shows myopia of right eye, but optic nerve normal.

The diagnosis was a cortical lesion (tumor ?) in the left hemisphere, involving the upper part of the motor area.

The following mixture was ordered: ℞. potassii iodidi, 15.; potassii bromidi, 30.; aquæ, 200.; S.: one teaspoonful before each meal, and two at bedtime, in plenty of water.

Oct. 10th. No spasm since beginning of the treatment, but the paralytic phenomena have increased; the walk is distinctly hemiplegic on the right side. Still works. Ordered to continue treatment, with addition of 4. ext. ergotæ fld. with the evening dose of bromide.

Oct. 13th. No attack. Speech normal; tongue deviates slightly to the right.

Oct. 17th. Slight spasm in the right arm yesterday; increasing paresis. Right hand squeezes 44° and 45°; the left, 50° and 45°. Ordered only three teaspoonfuls of bromide mixture at bed-time. To take besides twenty drops of a saturated solution of iodide of potassium three times a day in water.

Nov. 10th. No spasm; paresis of right leg more marked; walk distinctly hemiplegic.

Nov. 20th. Dr. Amidon was summoned to see the patient at his house. Has violent headache, more to the left of the median line at the vertex; photophobia, nausea, and almost constant vomiting. There is complete paralysis of the right arm and leg, and these parts are œdematous. Partial relief by hypodermic injection of .02 sulphate of morphia thrice during the day.

Nov. 22d. The pain has continued intense. Has asked to be killed. No aphasia. Eyes, examined by ophthalmoscope, show myopia 3.5 in each eye; fundus normal; sleep induced by rectal injection of chloral.

Nov. 30th. Headache has continued intense, requiring chloral and morphia. Has also had bromide and iodide of potassium as above. Some motion in fingers and right foot (lost on 31st).

Nov. 14th. Less headache, but continued right hemiplegia. Bed-sore beginning over sacrum. Some hesitancy of speech. At no time any aphasic defect.

Nov. 19th. Eyes again examined (without atropine); right fundus well seen, and found normal.

Nov. 21st. First signs of paresis in face; right cheek looks weak, and tongue points a little to the right side. Still has very severe headache.

Nov. 30th. Involuntary escape of urine. Scarcely able to speak from difficulty of articulation. At times silly.

Dec. 4th. Cannot be understood. Some contracture at right elbow, and the muscles of right arm and leg show some atrophy.

Dec. 19th. Paralysis now very marked about right cheek.

Dec. 31st. Quite a large bed-sore has formed on the right side of the sacrum. Marked atrophy of right arm and leg; elbow very stiff. Is semi-comatose. Pupils moderately small. Understands what is said to him, and tries to protrude his tongue when asked. Profuse sweating.

Jan. 2, 1880. Much brighter; speech can be understood. Of late has had no treatment except chloral occasionally.

Jan. 4th. Beginning of terminal stage. Fever and rapid respiration. A.M., axillary temperature, 38.8° C. At 5 P.M., asleep and sweating profusely. Pulse, 126; respiration, 26; temperature, 39.2° C. in axilla; in the rectum the thermometer indicates 40.1° C.

Jan. 5th. Fever and rapid respiration all night. At 11 A.M., pulse, 126; respiration, 56 (shallow); rectal temperature, 41.25° C. At 2 P.M., comatose without stertor; skin moist. Eyes in conjugate deviation to the right side; head straight. Pulse, 135; respiration, 50; rectal temperature, 41.6° C. At 4 P.M., died.

The autopsy was made by Drs. R. W. Amidon and W. R. Birdsall twenty-four hours after death. The calvarium was found very thin; translucent in spots. Dura mater normal. No subarachnoid fluid. There were many large superficial cerebral veins. The left motor area gave a sense of fluctuation; the convolutions of this part seemed normal, but were flat. On attempting to remove the falx cerebri in the usual manner, it was found adherent to the inner surface of the left hemisphere, pretty well back toward the tentorium. The cortex was ruptured in this location, and a gelatinous, bloody mass escaped. The rest of the encephalon seemed normal to external inspection.

A vertical transverse section was made through both hemispheres in the motor area, passing through the ascending frontal gyri. Occupying the centrum ovale underneath the left cortical motor area, and completely undermining it, was a large cavity capable of holding 100 cc. (?), very much resembling a distended lateral ventricle, which contained a large amount of coffee-red serum, and also a mass (tumor) lying on its inner side, near the paracentral lobule. The tumor was gelatinous and grayish-red. The walls of the sac were vascular and grayish, and appeared covered by an ependyma-like membrane, which, under the microscope, was found to consist of capillaries and portions of blood pigment.

The tumor itself had formed a connection with the falx cerebri posteriorly, in the region of the paracentral lobule, and this region of the cortex was thinned; it bulged across the median line and indented the opposite hemisphere.

On the left side the corpus callosum was pressed downward, and the optic thalamus was also depressed and flattened. The left lateral ventricle was displaced downward and closed by pressure; on opening it, it was found free from disease. These appearances were sketched from the fresh surface of section by Dr. Amidon, and are shown in Fig 3.

Sections made through the hardened brain confirmed the above notions of the seat of the tumor. It lay wholly beneath the externally visible convolutions of the left hemisphere, springing from and destroying that part of the first frontal gyrus which lies within the longitudinal fissure, above the corpus callosum and the paracentral lobule, forcing downward the gyrus fornicatus, extending outward into the white substance of the hemisphere, causing great compression of the surrounding parts, including the upper extremities of the first and second frontal gyri, the upper half of the ascending frontal and parietal gyri, and, to a less extent, of the upper parietal lobule.

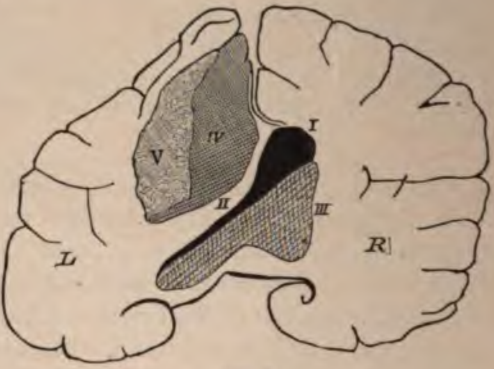

FIG. 3.

Transverse vertical section of the brain; Case II, viewed from behind. *R*, normal right hemisphere; *L*, diseased left hemisphere; *I*, distorted lateral ventricles; *II*, depressed corpus callosum; *III*, thalami optici, depressed on left side; *IV*, the tumor; *V*, the cavity formed by the hemorrhage.

A part of this pressure was due to the cyst lying outside of the tumor, near the convexity convolutions, which is more especially shown in the sketch made by Dr. Amidon from the fresh specimens.

The situation and dimensions of the lesion in this second case were, therefore, very different from those in the first case.

In Case II the destructive effects of the tumor were expended upon the gray and white substances lying next the longitudinal

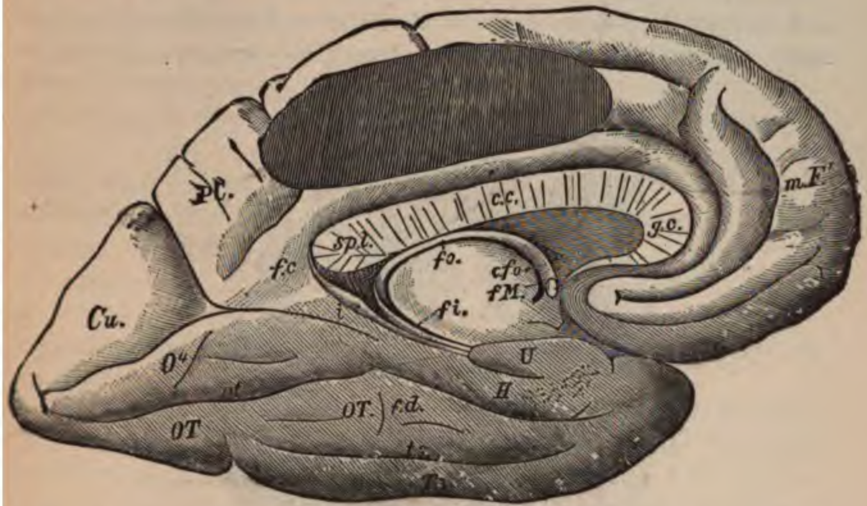

FIG. 4.

View of inner surface of the left hemisphere, after Schwalbe. Shaded spot indicates the superficial location of the tumor.

fissure, and the rest of the hemisphere suffered only compression effects. The posterior extremity of the intra-fissural part of the first frontal convolution and the paracentral lobule suffered the most destructive effects.

The tumor and cyst were of very irregular shape, and I can only give approximate measurements. In the longitudinal fissure and near it in the brain the tumor was about 60 mm. in length (antero-posterior dimensions); on a vertical transverse section of the hemisphere, as in Fig. 5, it measured 30 mm. transversely, and from 30 to 35 mm. vertically. These figures include the cyst, which was more developed in the frontal lobe, extending forward as far as the posterior part of the second frontal gyrus (wholly under it). The other (posterior) extremity of the lesion, the solid growth, could be traced, on the median surface of the hemisphere, well into the surface of the precuneus.

A microscopical examination of the tumor showed it to be a common small-celled sarcoma.

Remarks.—There are many interesting features in the semeiology of these two cases, but I shall dwell only upon those symptoms which are concerned in the questions of cerebral localization.

In both cases the first motor symptoms were epileptiform, and in Case II. the spasm was the first and only symptom for many months. In Case I. it was preceded by severe pain in the occipital region. In Case I. attacks of jerking of the left arm, as well as trembling of that member, were observed by the patient some weeks before the weakness became apparent. There was no jerking of the cheek or leg. It was a brachial monospasm. It is remarkable and most instructive to note how quickly paresis and paralysis followed, these phenomena being for a long time limited to the arm; a brachial monoplegia succeeding the brachial monospasm. Contracture of the arm and hand also showed itself, but at what time is not definitely stated.

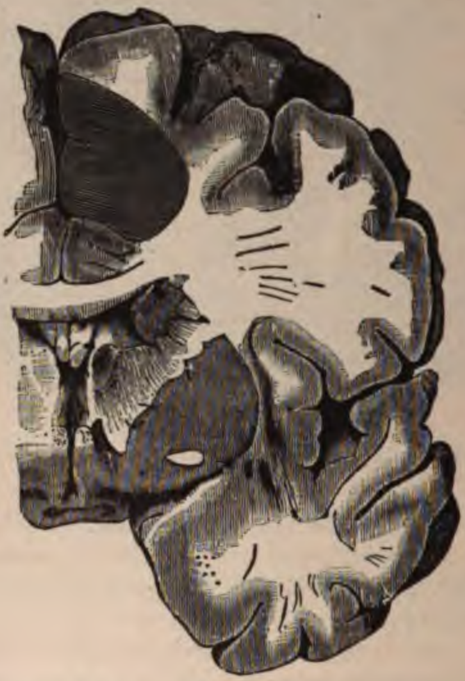

FIG. 5.

Transverse vertical section of left hemisphere, anterior view; after photograph No. 4 of Bitot Shaded spot in upper part of drawing shows the location of the solid tumor.

Late in the disease, when she came under observation, the left lower face and left third nerve were somewhat paretic, the sensibility was somewhat impaired on the left side of the body.

If it be permissible to formulate the chief symptoms observed during life in correlation with the lesion found *post mortem*, then this (Case I.) was a remarkable instance of irritating and destructive lesion of the upper part of the right ascending frontal gyrus, causing brachial monospasm and brachial monoplegia on the left side (with other phenomena of secondary logical value).

In Case II. the course of the motor phenomena was quite different. There was a period of two years, previous to the patient being seen by me, in which the only symptom was right hemi-epilepsy. That is to say, from time to time clonic, epileptoid spasms occurred in the right arm and leg for a few moments. The face was never affected, the patient could usually talk in the paroxysm, and he only once lost his consciousness. He was unable to say whether the spasm appeared first in the arm or in the leg.

At the time when the patient presented himself at the hospital the paralytic phenomena were just developing. He was still working all day at his trade, and was not conscious of the partial hemiplegia. This was, and remained until the apoplectic attack, more marked in the lower than in the upper extremity. At the time of first examination the right hand (affected side) was still stronger than the left hand, but the walk was slightly hemiplegic, the right foot being held in a slight equino-varus position. There was then no facial paresis and no aphasia.

Later the epileptiform attacks were controlled by bromide of potassium, but the hemiplegia progressed, still greater paresis being noted in the lower extremity.

About six weeks after first calling at the hospital, the patient was stricken down by an apoplectic attack, which rendered the right hemiplegia complete in the arm and leg, with marked paresis of cheek, but never aphasia. This attack obscured the symptoms which we may reasonably assume had been caused by the tumor. At no time was there marked anæsthesia on the paralyzed side.

Headache was remarkably slight prior to the occurrence of the apoplectic attack.

The *post-mortem* findings explain all these symptoms very well, I think. The cyst outside of the tumor proper, found in the white substance of the hemisphere, was the remains of a hemorrhage which took place at the moment of the apoplectic attack, which was characterized by intense pain in the head, vomiting, collapse, and complete right hemiplegia. Dr. Amidon states that in removing the brain a small laceration occurred, and "a gelatinous bloody mass escaped," probably the contents of the cyst, about seven weeks old.

The long stage of hemi-epilepsy without paresis, two years, is accounted for by the fact that the morbid growth began upon the median surface of the hemisphere, springing from the pia covering the inner winding of the first frontal gyrus, and perhaps the paracentral lobule; at any rate, for a long time it was an *irritating lesion* causing discharges, and only gradually exercised enough pressure to *destroy* the irritability of the neighboring gyri. The parts of the hemisphere which must have suffered first in a *destructive* manner were the paracentral lobule and adjacent parts (posterior extremity of first frontal gyrus on median surface), and in connection with this should be noted the fact that paresis of the leg preceded and preponderated over that of the arm, until the apoplectic attack occurred. The absence of aphasia throughout, and of facial paresis previous to the hemorrhage, are likewise of interest.

If I may venture to formulate this case, I should define it as one of *irritating and destructive* lesion of the left paracentral lobule (and adjacent parts), causing crural and brachial monospasm and monoplegia, with greater development of symptoms in the leg.

It will be seen by a reference to the now numerous recorded cases of localized cerebral lesions that the two cases which I report are in sufficient harmony with the results reached by many observers thus far, viz., that the "centres" for the hand and arm are in or about the ascending frontal gyrus in its middle region, while the "centres" for movements of the lower extremity are further backward in the posterior extremity of the ascending frontal and ascending parietal gyri, and their prolongation upon the median surface of the hemisphere, known as the paracentral lobule.

The many other interesting features of these two cases of cerebral tumor, I purpose considering in a future article upon the semeiology of cerebral tumors in general.

Before closing this contribution, it may perhaps be well if I present a brief *résumé* of the other cases of localized cerebral lesion which I have thoroughly studied (*i. e.*, while alive and *post mortem*) since the publication of my first paper on localization. Most of these cases have been published in medical journals.

CASE III.—Hemiplegia with first symptoms in foot, and a limited cortical lesion.

In November, 1878, I saw, in consultation with Dr. Granniss, of Saybrook, Ct., a gentleman aged 54 years, who was hemiplegic on the left side, and almost unconscious. The following account of his illness was furnished :

In December, 1877, after having enjoyed good health, he awoke one night with clonic convulsions of the left toes, foot and leg only. There was no impairment of consciousness, no spasm in any other part. He watched the spasm some time, and made comments on it. Since, there has gradually developed a left-sided hemiplegia. For months only the foot and leg were paretic ; in the last few weeks the left arm has become weak, and now the left cheek is paretic, though the relatives have not noticed it. In January, 1878, vision became impaired, but an examination by Dr. Noyes revealed no cause. In the last few weeks patient has seen double at times, and sight has gradually failed. Severe headache has existed from the first ; frontal, bilateral pain, most marked on the right side. The pain has been worst about daylight. In the past month pain decidedly nocturnal. On a number of occasions "lost himself" while out of doors, not remembering where he had been (petit-mal ?). A business associate thinks that patient has committed errors in judgment. No extravagance in design or in deed. Lately has become stupid and semi-comatose.

Since January, 1878, a tumor-like swelling has appeared over the right parietal region. No albuminuria, but has had several attacks of gout. After severe cross-examination, patient admits having had a chancre fifteen years ago, treated with mercury ; denies secondary and tertiary symptoms.

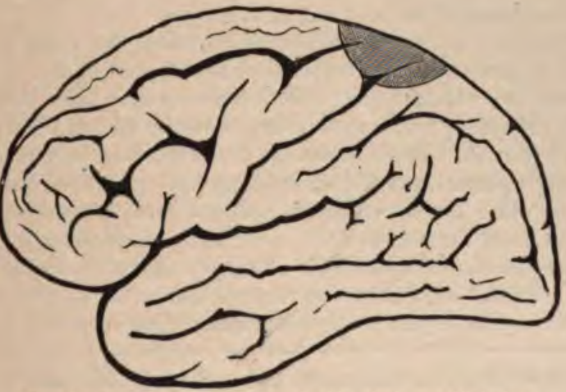

FIG. 6.—Lateral view of right cerebral hemisphere, with lesion.

Examination showed a typical left hemiplegia, face and limbs. No diplopia, pupils small and equal ; after atropia there is found a well-marked double neuro-retinitis. Sensibility preserved on the paralyzed side. Articulation indistinct, no aphasia. Stupor is peculiar, like that of drunken sleep. Patient can be roused by loud talking and shaking, and then answers correctly (showing fair memory) and clearly. The swelling upon the head, raised perhaps 1.5 cm., is just above the right parietal eminence, extending inward to the median line, and forward almost to the vertical line from the meatus auditorius to the bregma. This tumor overlies Ferrier's centres for the leg.

Diagnosis : External and internal nodes involving dura mater and the subjacent gyri of the right hemisphere.

A few days later the patient died comatose, and after much trouble Dr. Granniss secured a partial autopsy. He was not allowed to raise the brain from the skull or to incise it. He simply removed the calvarium and noted the lesions at the vertex. He found that there was an internal as well as an external osteitis, forming quite a tumor, which had, after adhering to the dura, exerted great pressure upon the subjacent convolutions of the right hemisphere. Dr. Granniss marked the location of the cortical lesion upon an Ecker's diagram, and the annexed wood-cut is a copy of his sketch.

It is of course very much to be regretted that a thorough examination of the brain was not permitted, but in view of numerous recent cases, it is impossible not to admit a causal relation between the lesion causing pressure upon the inner end of the right ascending frontal and parietal convolutions and the symptoms in the left foot and leg—spasm and paralysis.*

CASE IV.—Aphasia with word-deafness; no permanent paralysis; lesion in the parietal region.†

The main facts of the last illness of the late Dr. C. M. A., of New York, are already well known to his numerous friends in the medical profession, who watched the progress of his disease with painful interest. Throughout his illness he was attended by his partner, Dr. A. Dubois, and myself. He was also seen in consultation by Profs. Austin Flint, Sr., John T. Metcalfe, H. D. Noyes, and Dr. Allan McLane Hamilton, and for several months was under the professional care of Prof. E. C. Seguin.

Dr. A. was born in 1827, and was therefore fifty-two years of age at the time of his aphasic attack. At the age of eleven years he had a long illness, which was called "brain fever." Whatever may have been its real nature the illness was sufficiently severe to seriously endanger life, and for several years retarded his growth. At about the age of thirty years he had an attack of inflammatory rheumatism affecting the larger joints. This was followed by three or four other attacks within the next few years, but none of them lasted longer than from three days to a week, or was attended, so far as we can learn, by any cardiac complication. Twelve years ago he had a well-marked attack of gout, and since then had three or four other paroxysms, the most severe one five years ago, after a violent quinzy, when both great toes were affected. For several years before his aphasic attack, he was subject to flatulent dyspepsia, and had occasional outbreaks of eczema. It should be noted here that neither gout nor rheumatism was hereditary in his family, and that the most frequent cause of gout—over-indulgence at the table—was notably absent in his case, as he was usually very abstemious both in eating and drinking. In November, 1877, he had a severe attack of renal colic. The concretion was arrested in the ureter, and not discharged until the end of ten days, after repeated paroxysms of colic. The stone, on analysis, was found to be composed of uric acid. On February 1, 1878, he attended a concert in evening dress, and on his way home became thoroughly chilled. During the night he was awakened by pain and oppression in the chest, these symptoms continuing during the following day. As there was no evidence of pulmonary lesion, but merely tenderness over the middle portions of the chest anteriorly, on both sides, with pain in these situations on movement of the pectoral muscles, the symptoms were referred to muscular rheumatism. Within a few days he was able to return to business, but was still so far from well that some more serious disturbance was apprehended by his medical attendants.

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* *Archives of Medicine*, vol. ii., p. 105. (A remarkable case of hemorrhage under the paracentral lobe, with paralysis of the opposite leg, is recorded by Dr. Miles, of Baltimore, in the same journal, p. 103.)

† Dr. A. B. Ball. A contribution to the study of aphasia, etc. *Archives of Medicine*, vol. v., No. 2, April, 1881, p. 136. Inserted here with the permission of Dr. Ball.—[R.W.A.]

On February 11th, the date of his aphasic attack, he was in much better spirits. At half-past eight in the evening he was seen in his office writing a letter. A few minutes before ten o'clock he rang his bell violently, and was found by his servant lying on the lounge talking unintelligibly. I saw him not more than five minutes afterward. He was conscious, but unable to answer questions except by a confused muttering. The face was slightly flushed; pulse soft, easily compressible, about 90 per minute; the first cardiac sound feeble, and no murmur audible. Incomplete right hemiplegia and right hemi-anæsthesia. Was apparently aware of the nature of his attack, as he pointed to his right arm and left frontal region. By gestures he finally succeeded in directing my attention to important cases in his note-book, requiring attention on the following day. At 11 o'clock he was seen by his partner, Dr. Dubois, and with slight assistance walked up two flights of stairs to his bedroom. On the following morning he complained of paroxysms of pain in the left frontal region. This symptom, which yielded to local applications of hot water, annoyed him frequently for several weeks, and recurred at intervals during the whole course of his illness. Repeated examinations of the heart failed to disclose any morbid condition except feeble action and moderate hypertrophy. No albumen or casts in the urine. Absence of fever, except on the evening of the third day, when there was a slight rise of temperature which lasted only a few hours. From this time his physical condition steadily improved, and by the end of six weeks his general health was fairly restored. Beyond slight paresis of motility and sensation on the right side the only marked change was the aphasic condition to be presently described. During the summer and autumn of 1879 his physical condition remained fairly good. The kidneys performed their work well, although it was evident from the occasional appearance of traces of albumen and casts in the urine, and from the enlargement of the left ventricle without valvular murmurs, that the kidneys had probably undergone cirrhotic changes. At no time was any increased arterial tension noticed in the sphygmographic tracings, but this absence was ascribed to muscular degeneration of the cardiac muscle, as feeble action of the heart was a constant symptom throughout his illness.

In March, 1880, he had another attack, which was supposed to be due to a small cerebral hemorrhage. At dinner, while talking with a friend, he suddenly turned his head to the right, and began muttering incoherently. With assistance, he immediately left the apartment and walked to his bedroom, muttering all the way with his head turned to the right. At my visit, half an hour later, when his consciousness was fully restored, he said that the attack began with an explosive noise in the head like a pistol-shot. Immediately he heard some one talking to him over his right shoulder, and turned to see who was addressing him.

Every word uttered by himself, he said, was mockingly repeated by this imaginary individual, and the mutterings his friends had heard were his indignant protests against the insult. On examination there was found slight paralysis, with numbness and anæsthesia on the *left* side. These symptoms disappeared after a few days, his mental condition remaining without apparent change. Shortly after this attack it was evident that his heart was failing in power. He frequently complained of breathlessness on exertion, and the heart sounds were feeble, with occasional intermittence of beat. Toward the end of May he was seized with what proved to be his final attack. The symptoms were slight fever for several days; oppression in the chest, with shortness of breath; slight cough, generally dry but occasionally accompanied by expectoration tinged with blood; and marked tenderness over the region of the heart. At a few examinations a faint aortic obstructive murmur was heard, or rather a soft blowing sound over the base of the heart near the aortic valves, with the first sound. Urine nearly normal in amount; specific gravity varying from 1,012 to 1,018; no albumen, and no casts except a few hyaline cylinders found at one examination. These symptoms were hardly sufficient to warrant a positive diagnosis, but they seemed to point to endocarditis with possibly myocarditis, and this view was confirmed, or at least considered plausible, by Prof. J. T. Metcalfe, who saw him in consultation. The urgent symptoms subsided by the end of a week, but he was still much prostrated, and complained of giddiness and mental confusion. On one occasion he exhibited in a marked form the so-called rotatory phenomenon, turning over rapidly to the right, and would have rolled out of bed had he not been prevented. On June 19th, about 3 P.M., he suddenly became totally blind. Dr. Dubois, who saw him shortly afterward, found him still partially blind, but gradually regaining his vision. At my visit, two hours later, he was perfectly conscious, with his sight fully restored. Half an hour afterward he fell into a quiet slumber, from which he suddenly awakened at 7 o'clock, exclaimed "Oh!" and died instantly.

In considering the aphasic symptoms which constituted the most striking and interesting feature of his case, a few preliminary remarks on the essential nature of aphasia may be permitted before analyzing the symptoms in detail.

The interchange of thought between members of the human family is carried on by means of various symbols, that is, by signs which stand for the ideas they represent; for example: articulate sounds, written language, gestures, facial expression, mathematical, musical, and other signs. In aphasia this symbolic function, or capacity to *interpret* and *express* thought in a symbolic form—the *facultas signatrix* of Kant—is more or less seriously impaired. In some cases the chief difficulty is in the direction of *symbol expression* (ataxic aphasia), the concept being present, but failing to enunciate itself on account of some lesion in the motor tract concerned in the expression of symbols. In other instances the concept is present in the mind, but the appropriate symbol for it is forgotten (amnesic aphasia). In a third class of cases there is also a defect in the capacity for *comprehending* symbols. Certain auditory and visual impressions, especially those of word symbols, fail to recall into consciousness their corresponding concepts, although the capacity for forming such concepts under the influence of other stimuli may still be retained. When concepts can no longer be formed, the lesion involves the fundamental processes of thought, and extends beyond the sphere of simple aphasia. The latter term fails, however, to recognize the impaired capacity to *understand* symbols, and as most cases of aphasia present some degree of this derangement, Finkelburg* has proposed to substitute the word "asymbolia" as a generic term for all the phenomena of aphasia. Kussmaul † prefers the term *asemia*, suggested by Steinthal, as being still more comprehensive; "symbol" represents an idea behind it, whereas "sign" often represents merely an emotion. In the following description of the aphasic symptoms in Dr. A.'s case, we shall use the word "symbol" in preference to "sign," as there was no difficulty in comprehending or expressing emotions. Our classification is based upon that of Spamer. ‡

I.—EXPRESSION OF SYMBOLS.

a. Disturbances of speech.—On the morning following the first paralytic seizure, by which time the general shock to the brain had abated, it was evident that the cerebral disturbance was limited chiefly to the *verbal* expression of ideas. His general intelligence was fairly well preserved, and he understood much that was said to him, but there was a marked defect in verbal expression. His principal difficulty was with proper names and common nouns. When a glass of milk was held before him, he said: "That is something to drink," recognizing at once its several attributes, its color, uses, etc., but the word which combined these qualities into a single concrete expression, or symbol, he could not utter, even when the word was repeated to him. He had less difficulty with adjectives, verbs, and adverbs, that is, with words of less concrete symbolic character. His vocabulary of proper and common nouns very soon began to increase. Within the first few days we succeeded in teaching him a number of such words by directing his attention to the movements of the lips and tongue in pronunciation. My own name, being short and easily pronounced, he learned in one day, and rarely afterward forgot it. Long names of individuals, or long words which he rarely had occasion to use, he seldom mastered completely at any period of his illness. During the summer and autumn of 1879 his vocabulary increased so as to include a considerable number of words used in ordinary conversation. With these he generally succeeded in expressing his ideas fairly well, but an attempt to leave the beaten track resulted in mental confusion and inability to proceed with the conversation. In rare instances his conversational powers astonished his friends, and gave him delusive hopes of ultimate recovery. On one occasion he conversed with fluency on various topics for nearly an hour, with a friend who had not met him for several years and was unaware of his illness. His friend noticed no aphasic disturbance during the interview, and was greatly surprised afterward on learning the facts of the case. Such flashes were, however, only intermittent, and it became more and more evident that anything like perfect recovery was hopeless.

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* *Berl. Klin. Wochenschrift*, 1870, band vii., p. 449, 460.

† *Ziemssen's Cyclopædia of Medicine*, American edition, vol. xiv., p. 609.

‡ C. Spamer. *Archiv für Psychiatrie*, band vi., p. 526.

In conversation, true *paraphasia*, that is, the substitution of wrong words, was rarely noticed. Almost invariably the word uttered bore some resemblance to the correct one, and differed from it in only some of its letters. Thus the first letters were usually correct. This fact was of great assistance to him in conversation, as it enabled him, when he knew the first letter, to find the correct word in a dictionary or work of reference, *where* *he at once recognized it as soon as he saw it*, showing that the concept was present in his mind in a latent form, and needed only the right stimulus to recall it into consciousness. His Medical Register was frequently consulted for physicians' names he was unable to pronounce, as he retained, to a marked degree, his interest in news affecting the medical profession.

In the expression of musical and other non-verbal sounds, as in singing, whistling, and imitation of various significant sounds, there was no observable deficiency.

As regards the *alphabet* and *numerals* the same cannot be said. At the outset of his illness he was able to pronounce only a few letters, and could not count above four. With training, however, he in time learned most of the alphabet, but never succeeded in spelling any but short and simple words. Counting he reacquired quite perfectly, and was able to solve simple sums in arithmetic, that is, to express their answers verbally. Even when unable to do this he could often *write* the answers correctly. When both these efforts failed him he was frequently able to recognize the correct answers if shown to him in writing. During the latter part of his illness he supervised his business accounts, and rarely failed to notice mistakes in them made by others. This circumstance belongs, however, rather under the head of symbol-*comprehension* than under that of symbol-*expression.*

b. Defects in writing.—At the outset of his illness there was complete *agraphia.* When asked to write the word "cat," he took the pencil in his left hand, and drew three perpendicular lines, naming them one, two, three. As we shall see in a later illustration, this substitution of numerals for letters and words was at first very noticeable. He knew the number of letters required for the word "cat," but there was *no attempt at the formation of letter symbols,* although he was perfectly aware that his straight lines were not letters. Under training he gradually learned to form letters with his right hand, and after several months could copy simple sentences correctly, sign his name in his usual clear and elegant handwriting, and even write short sentences of his own composition, but more than this he never succeeded in accomplishing.

c. Gesture language.—The capacity for expressing ideas by gestures seemed to be unimpaired. He retained much of his natural vivacity of manner, more in fact than could have been expected in a person of his keen sensibility, when he found himself cut off from the ordinary modes of social intercourse. His gesture language had always been a prominent characteristic, and now became an important aid in the expression of ideas. Names of individuals and objects, which he was unable to remember or to pronounce, he frequently succeeded in recalling to others by gestural description, and this was very noticeable even early in his aphasic attack.

II. COMPREHENSION OF SYMBOLS.

Before entering upon this branch of our subject it should be noted that the senses of sight and hearing in the present case were perfect, so far as could be determined by the usual tests. With respect to vision, the only exceptions to this statement were a transient attack of total blindness a few hours before death, and occasional attacks of hemiopia. Prof. H. D. Noyes, who made an ophthalmoscopic examination of his eyes in the autumn of 1879, reports that "he found no remarkable change in the optic nerves or retinæ. The arteries of the nerves were rather small, and, with this exception, nothing abnormal was noted."

A.—*Comprehension of Auditory Symbols.*

a. Spoken words.—Early in his illness, on my remarking to him one day, "Dr. Peters called to see you," he replied, "I don't know him." The name was repeated several times, but he failed to recognize it, although it was the name of an intimate friend. The written name was then shown him. "What a fool I am," he exclaimed; "of course I know him." This was the first instance in which my attention was drawn to the fact that certain auditory impressions failed to be converted into concepts, although the conceptive faculty remained intact. Not long afterward he noticed this peculiarity himself, as was shown by his remarking to me: "The words I can't pronounce are the words I can't *hear.*" This observation, the general correctness of which was verified by repeated experiments, points to a very interesting peculiarity in his case. The words over which he stumbled in conversation were words which made no intelligible impression on his mind when repeated to him, and, conversely, the words he failed to understand in conversation were words he had great difficulty in pronouncing spontaneously. The concepts represented by these word symbols we were generally able to recall to his consciousness by other means, such as writing, gestures, etc., but even then he was unable to express them, except after a certain amount of training. This "word-deafness," except when it was possible to stimulate the conceptual centres by visual or other impressions, made it extremely difficult to determine how much of his aphasia was due to the *ataxic* and how much to the *amnesic* element.

b. Musical and other sounds.—His appreciation of music was fortunately well preserved, and was a source of much pleasure to him. In attending concerts and operas he exhibited his usual good critical taste. The significance of other sounds, such as the tone of a bell, the striking of a clock, etc., was perfectly understood.

B.—*Comprehension of Visual Symbols.*

On the third day of his aphasic attack a scroll of Scripture texts was held before him, and he was asked to read the following sentence: "We love Him because He first loved us. While we were yet sinners Christ died for us." He read aloud as follows: "We he have two three that I have to have the same. I have two three." The substitution of numerals for words is here again noticed, as in a previous illustration. The words "the same" probably refer to the repetition of "love" in the first sentence. He was aware that this rendering of the text was incorrect; in fact he almost always knew when he read aloud incorrectly, and expressed impatience thereat. Later in his illness, when he was able to read sufficiently well to gather from the newspaper the main points of news, he remarked to me that there were always words in every long sentence which conveyed no impression to his mind, and that he was compelled to form his idea of the meaning of such a sentence from the other words whose meaning he understood. The significance of many of these uncomprehended words could be conveyed to him in other ways, showing that his failure to recognize the written symbols was not always due to a defect in the conceptual centre, but rather to a lesion in the channel of transmission from the optical centre for word symbols to the ideational centres.

The same difficulty extended at first also to the comprehension of *written numerals and their combinations*, but, as we have already seen, he reacquired, to a certain extent, this capacity under training. *Gesture language* he understood perfectly from the start.

The degree of impairment in intelligence, otherwise than in the comprehension and expression of symbols, it was extremely difficult to determine, for reasons already given. His intimate friends were satisfied that there was much less general mental deterioration than those who met him casually would infer. His memory of incidents in his own life, of the past illness of his patients, and of numerous other details, was strictly accurate, so that we could rely upon his statements upon such points in every particular. In business matters he always manifested his usual tact and good judgment. During the last few months of his life he was a constant attendant at the surgical operations of the New York Hospital, of which he was an attending surgeon, and his criticisms showed that he retained not merely a general interest, but also his special knowledge in surgery. On several occasions he assisted me in minor surgical operations and dressings, with his usual deftness and attention to details. At whist, euchre, and all games with which he had been familiar, he was as expert as ever. During the winter of 1879–80 he consulted numerous medical works on the subject of aphasia. Since his death I have seen a sheet of paper containing his notes of reference to articles on this subject in English and French works and journals. The titles, dates, etc., are strictly correct, and are written in his usual clear and elegant handwriting. His memory of location was particularly well preserved. He could always turn without hesitation to the right place in books he wished to consult, remembered the houses of friends—that is, their relative positions in this city,—and in numerous other ways showed that he perfectly understood the spatial relations of objects. The only exception to this fact was a singular symptom which annoyed him for several months, viz.: a tendency to reverse the natural position of objects which he handled, such as table-knives, spoons, pencils, canes, etc. He immediately recognized his mistake, however, and corrected it, but always spoke of the inclination as irresistible.

As an aid to the interpretation of the aphasic symptoms in the present case, we reproduce, below, Spamer's diagram, representing the several tracts between the reception of impressions, the comprehension of these impressions, and their expression.

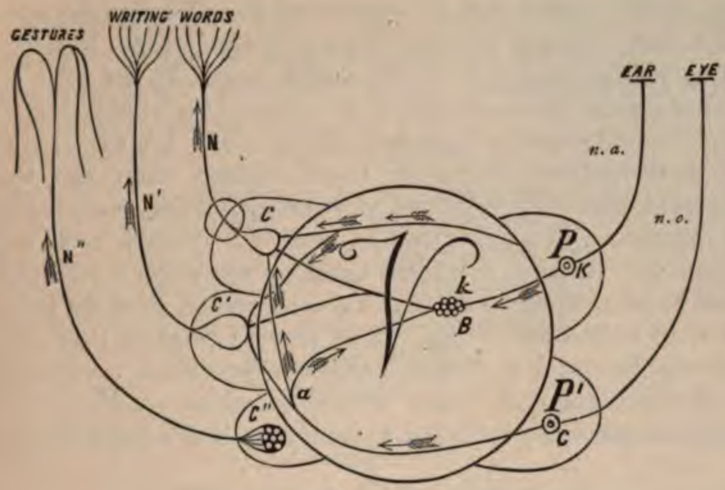

FIG. 7.

The circle in the middle of the diagram, *V*, represents the ideational tracts. From the right the excitations of the sensory nerves pass into the brain.

n. a.—auditory nerve. *n. o.*—optic nerve.

P and *P'* represents the places where the auditory (*K*) and the optical (*G*) impressions are perceived. When the impressions reach these points we have merely sense-perceptions without associated conceptions. The association with definite corresponding conceptions takes place only when the excitation travels onward to *B*, the conception. From this point the excitation may proceed to *C*, *C'*, *C''*, the centres of co-ordination for movements in speech, writing and gestures.

N, *N'* and *N''* are the motor nerves concerned in symbol expression (speech, writing, gestures). At their termination these nerves are broken up into fibres distributed to individual muscles.

The diagram represents the reception and tracts of *word symbols* through the eye and ear. The tracts of other auditory and ocular impressions are not designated.

It will be noticed in the above diagram that the tract from *P* to *B* is represented by a straight line, while the tract from *P'* to *B* pursues a circuitous route. By this distinction Spamer attempts a rough explanation of the difference observed in most cases of aphasia between the comprehension of *auditory* word symbols and the comprehension of *visual* word symbols. Cases of marked word-deafness, without ordinary deafness, seem to be extremely rare; at least there are very few instances of this kind on record. The tract for *all* auditory impressions, he supposes, lies in close connection, and may be represented by a single straight line. With visual impressions the case is different. Aphasic patients very generally recognize material objects, but exhibit a marked defect in understanding written and printed words, as well as in expressing the concepts in speech and writing. The tract for visual word symbols is, therefore, more or less widely separated from the tract for other visual impressions, and lies in some parts of its course near the centres of co-ordination for speech and writing, or near the tract from *B* to the latter. This explanation is ingenious, but hardly satisfactory. If the tract from *P'* to *B* should be represented by a circuitous route, that from *P* to *B* could scarcely have been *direct* in Dr. A.'s case, because the word-deafness was even more marked than the word-blindness, although both auditory and visual impressions, with the exception of word symbols, were interpreted with equal acuteness. Indeed, our main reliance, when the word symbol failed to be recognized by him in conversation, was to present the word to him in writing. The reverse process, that is, the presentation of the auditory, in place of the visual, word symbol rarely succeeded. In other words, he seldom understood the spoken words when he failed to comprehend the written form.

DR. SEGUIN'S REPORT OF THE AUTOPSY.

The autopsy was made twenty hours after death, on June 20th. The body was well preserved in ice.

Head.—The dura mater is abnormally adherent to the calvarium, on both sides equally; no thickening of dura. Pacchionian bodies small. Marked subarachnoid effusion, which has gravitated to posterior regions. Dura of base normal. The basilar artery is really a continuation of the right vertebral artery; the left being only 1 mm. thick. The right vertebral and the basilar arteries are the seat of patches of arteritis, separated by regions of healthy tissue, but nowhere obstructing the flow of blood. Circle of Willis is complete and patent. The carotids, just below the circle of Willis, are extraordinarily thickened, quite rigid, but not calcareous; their wall is nearly 1 mm. thick. The same alterations in patches can be traced in the accessible branches of the middle cerebral arteries; the anterior cerebrals are only slightly affected. Nerves at the base normal. The left hemisphere is the seat of a large depression caused by the destruction of several convolutions, viz.: the whole of the inferior parietal lobule, with the first tier of temporal gyri. The posterior extremity of the angular gyrus, and the whole of the ascending parietal, are preserved. This lesion is a yellow patch lying in the region supplied by the terminal branches of the left middle cerebral artery. To external examination, the remaining convolutions are normal, more especially the third frontal, the ascending frontal, and the anterior gyri of the island of Reil. The first branch of the middle cerebral artery on the left side is pervious, though there are a few patches of arteritis near its origin. The main trunk of the artery, in the fissure of Sylvius, and its two terminal branches are pervious to the confines of the patch, and in the pia covering the patch. The patch was probably caused by blockade of

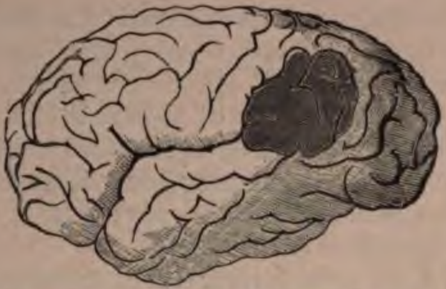

FIG. 8.—Lateral view of left cerebral hemisphere, after Henle. Shaded spot shows the superficial location of the yellow patch.

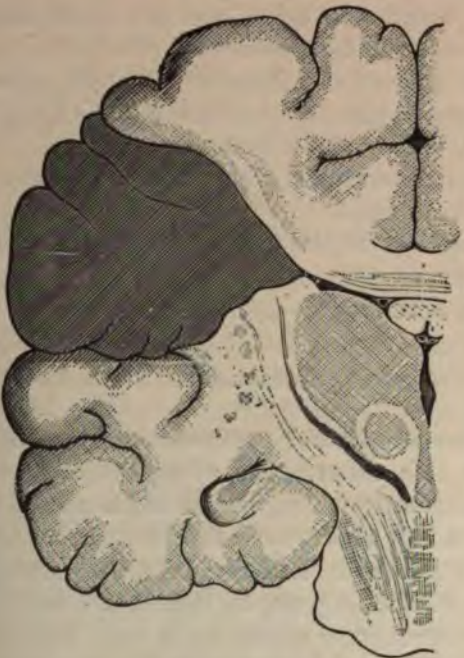

FIG. 9.—Diagram of transverse vertical section through left hemisphere, showing the extension inward of the patch. This view corresponds to section No. 4, described in the text.

smaller arteries which cannot be traced. The right hemisphere presents a healthy surface. On opening the fissure of Sylvius, the middle cerebral artery is found patent, but bearing a few patches of thickening.

The brain is sliced in transverse vertical sections.

Section No. 1, about 37 mm. from apex of frontal lobes, presents no lesion.

Section No. 2, at a distance of 25 mm. behind No. 1, passing through the posterior extremity of the third frontal convolution and cutting off the apex of the temporal lobe, is free from lesion.

Numerous fine slices made in the speech tract in this region (left side) reveal no alterations of structure.

Section No. 3, 25 mm. further back, showing the lenticular ganglion and the thalamus, no lesion.

Section No. 4, made at 25 mm. behind No. 3, passing through the anterior limit of the yellow patch above described, and cutting through the posterior extremity of the thalami. There is no lesion to be seen except the yellow patch in the left hemisphere, and its full extent is well shown; besides destroying the convolutions it extends deep into the white substance of the hemisphere to the roof of the lateral ventricle.

Section No. 5, made at a distance of 25 mm. posterior to No. 4, reveals the penetration of the yellow patch as just described.

Section No. 6 shows no lesion.

The brain was afterward finely sliced up without any other lesion being discovered.

Sections made at different points in the pons Varolii and medulla oblongata seem normal.

Cerebellum normal.

CONCLUDING OBSERVATIONS BY DR. SEGUIN.

Dr. A.'s paræsthesiæ and perversions of muscular sense were very curious. He referred his sensations of numbness on the right side to homologous regions in the hand and foot, viz.: the distribution of the ulnar nerve and that of the musculo-cutaneous in the leg and foot. In the right side, generally, the paræsthesiæ were of drawing up, or tightening, and as if a strong rotatory movement were going on in each limb around its longitudinal axis, the hand in pronation, the foot in inversion. The patient's account of these subjective movements never varied, and he would often illustrate them by moving his hand and forearm in extreme pronation and rotation.

The impairment of muscular sense of which he complained was something which I had never met with before. If he did not use his eyes in prehending objects with his right hand, he would find that he had seized them by the wrong end. He sometimes found himself standing with the head of his cane on the ground and its point in his hand. Frequently, in my presence, he essayed to grasp a pen or pencil with his head turned away, and repeatedly he found himself holding the object by the wrong end, and this after turning it over three or four times to get its outlines.

Yet with these perversions of sensibility there was no common anæsthesia, either to pricking, to cold, or to æsthesiometer points.

Dr. A.'s aphasia was complex, but the striking feature in it, during my six months' observation, was the word-deafness.

He could express himself fairly well in short sentences, and might for a little while carry on a commonplace conversation with a non-expert without betraying his defect; but he frequently failed to find the right word, and often found it only after struggling a good deal.

In attempting to speak he would often, after failing to get the proper noun, use a corresponding verb or employ synonyms, showing that his idea or concept was always correct, but that his vocabulary was faulty. He could copy written or printed characters quite readily, but experienced great difficulty in writing spontaneously.

All the auditory relations of language were much impaired. He used to say that going to church and listening to a sermon was to him all a mixed-up, meaningless jargon, like "drub-arub-drub." He could catch very few words. In ordinary conversation, familiar short sentences were apprehended readily; equally simple sentences, containing other than the most commonplace words, had to be repeated again and again. Reading from a book was jargon to him. Writing from dictation was impossible, and even the alphabet was poorly executed in this way. The sound of the letter *c* seemed the one for which he was most deaf.

Yet his hearing was not impaired (I never tested it carefully), and he understood and appreciated music. While a lecture or a sermon was unintelligible, he enjoyed a concert and claimed to appreciate it. He whistled and hummed airs correctly—much better than he spoke.

I often questioned about and tested him for hemiopia, with negative results. Occasionally he had attacks of moving fortification lines in the left fields of vision, but these were evidently phenomena of the migraine type.

The pathology of the case is obscure in many respects.

The arteritis (see Dr. Peabody's description) is not of the senile type, and the patient's statement that he had never had syphilis was positive, and, we believe, perfectly trustworthy. This would, therefore, be one of the best authenticated instances of non-specific endarteritis deformans, leading to obliteration of the calibre of small arteries, ischæmia of a cerebral territory, and softening.*

The location of the lesion is peculiar, and some years ago would have been considered as destructive of the modern theory of aphasia. In view of the experiments of Ferrier, Munk,† and others, however, it seems clear that the lesion occupied a portion of the brain which is concerned in the reception of sensory impressions from various sources, more especially the eye and ear.

So long as aphasia was looked upon as sometimes a form of motor disorder, a difficulty in the emission of language, and in other cases as dependent upon verbal amnesia, it was impossible to explain its production by a lesion of the parietal or sphenoidal lobes. In the last two or three years the elements of imperfect perception of the written signs and spoken sounds of language—word-blindness and word-deafness respectively—have received some recognition, and these phenomena are perfectly explicable by lesions placed in the sensory or perceptive regions of the cortex and internal capsule.

In such cases the aphasia is indirect, not due to any interference with the channel for the emission of sound-forming impulses, but to a break in the other part of the circuit, viz., the receptive organ.

Dr. A. B. Ball, of New York, is the author of the article from which the foregoing large extract is made, and my small share in it is the description of the lesion found in the brain, and some general remarks upon the pathology of aphasia. In this connection I quote from the article because the lesion seems to indicate the postero-inferior limit of the motor area of the hemisphere. Although a large part of the inferior parietal lobule, and the first tier of temporal gyri, together with the associated white matter, were necrosed, there was no permanent hemiplegia. At the beginning of the illness, for awhile after the attack, "slight paresis of motility" was noted. Whatever value this case may have for the study of indirect aphasia, it certainly will rank high as a negative case in the question of cortical motor localizations.

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* It is very much to be regretted that the cerebral arteries and the brain itself were not examined microscopically; but the autopsy was allowed only on condition that the brain be not retained for examination.

† Consult Ferrier, The Functions of the Brain; New York, 1876; Munk, Ueber die Functionen der Grosshirnrinde; Berlin, 1881.

CASE V.—Abscess of the left frontal lobe of the cerebrum, without motor phenomena.*

On April 11, 1880, I was asked by Dr. J. Lewis Smith to see a case in consultation with himself and Dr. J. R. Leaming. The patient was a young married woman, aged about 28 years, who had formerly enjoyed good health and had borne several children. During the month of February one of these children had died after a severe illness, and she had undergone considerable fatigue. She seemed depressed, weak, and anæmic afterward.

About four weeks before the date of consultation she complained of pain over the left eye. This was soon accompanied by swelling and exophthalmus, and on March 24th Dr. Knapp was called in and diagnosticated orbital (subperiosteal) abscess. This was opened on March 26th by Dr. Knapp.

It was remarked that the pus was under great tension, and that it spurted out a considerable distance when released. Pain ceased at once, the exophthalmus disappeared, and the wound quickly healed. During the first few days of April all seemed going on well; the wound was healed; the patient was free from pain; she was taking tonics, and on the 3d made a call on a near neighbor.

During the night of April 3d and 4th, one week before my examination, she awoke with severe headache and vomiting; ever since she has lain abed, presenting the following symptoms: Headache, chiefly mastoid and through the base of the skull; occasional vomiting; irregular respiration; irregular and very slow pulse, varying from 60 to 50 beats per minute; stupor and general feebleness. As negative points there were no symptoms about the eyes, objective or subjective, except a partial ptosis of the left upper lid (which had been incised); no fever, chills, convulsions, paralysis, aphasia; at no time had there been coma. The urine was free from albumen.

Examination.—Patient was soporose, but could be roused by loud speaking; she answered questions as if half asleep, but in such a way as to leave no doubt as to the preservation of language. She put up both hands to the mastoid regions when indicating the seat of pain. A minute inspection showed no paralysis except about the left eye, whose upper lid drooped and whose internal rectus was inert. The pupil on the left side was not fully dilated, but it was a little wider than the right. The optic nerves appeared somewhat congested, and were dim at their periphery, but there was no actual choking. Patient appeared to feel pinching well everywhere. The thermometer showed no fever. The pulse varied from 53 to 66 beats per minute, and it was a reluctant, delusively full pulse, with no real strength. The breathing was easy and regular, but friends of the patient described quite well a Cheyne-Stokes breathing which they had observed. There was neither redness nor tenderness about the site of the orbital abscess.

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* See p. 452; also *Archives of Medicine*, vol. v., No. 1, Feb., 1881, p. 107.

I diagnosticated an abscess of the brain, probably in the left frontal lobe, and expressed the opinion that the patient was in imminent danger. She died the next day in a comatose state, no new symptoms having been observed.

It was then learned that for two years Mrs. F. had suffered from frequent attacks of headache, lasting several hours. The pain was frontal, and sometimes extended along the nose and into the left temple. There had never been symptoms of chronic nasal catarrh.

The autopsy was made by me on April 13th, about thirty hours *post-mortem*, in the presence of Drs. Knapp, J. R. Leaming, J. Lewis Smith (the attending physician), and Richard Wiener. We found a large abscess, the size of an English walnut, in the left frontal lobe. It seemed to lie wholly under the cortex cerebri, in the convolutions of the orbital lobule, and in the second frontal convolution. Viewing the hemisphere from

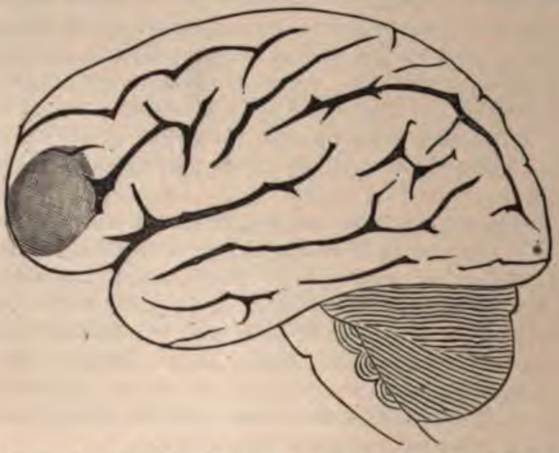

FIG. 10.—Apparent location of the abscess, drawn on an Ecker's diagram of the brain.

the side, the apparent posterior limit of the abscess was the anterior border of the lower part of the third frontal gyrus. Fig. 10 indicates the seat of the soft, fluctuating, bulging abscess. Its size and penetration were not then determined, as it was thought best to harden the brain as a whole before making sections.

The external connections and origin of the abscess were most interesting. There was only one point of adherence between the diseased frontal lobe and the dura mater, and that was over the orbital plate of the frontal bone immediately under the swollen frontal lobe. There the dura mater was thickened and adherent to the pia mater and cortex cerebri, forming the inferior wall of the abscess, over a space as large as a ten-cent piece (about 15 mm.). Under this patch of pachymeningitis the orbital plate of the frontal bone was necrosed and perforated; a probe was easily passed into the orbit.

In the orbit, under its periosteum, pus was found, and a part of the roof and the inner wall of the orbit were carious. Careful dissection by Dr. H. Knapp showed disease of a similar kind in the ethmoidal cells and frontal sinus. I need say nothing more of the conditions of these parts and of the pathology of the orbital abscess, as the case has been fully reported from this point of view by Dr. Knapp.*

The appearance of the necrosed orbital plate and of the thickened, adherent dura mater, was precisely similar to what I have several times seen in cases of suppurative disease of the internal ear with cerebral abscess by contiguity. The genesis of the abscesses must have been alike in the two situations.

In December, the brain having been sufficiently hardened in bichromate of potash solution, I embedded it in Gudden's microtome, and made several horizontal sections through the whole brain with the view of demonstrating the relations of the abscess. These cuts showed that the abscess was of quite as large a size as at first supposed, almost perfectly globular in shape, measuring about 38 mm. in diameter. It contained ordinary pus, and was lined by a distinct membrane 1-2 mm. thick. The anterior, inferior, and external limits of the abscess were thinned cortex and pia mater; superiorly, posteriorly, and internally, it was bounded by apparently normal white substance. The whole of the white centre of the frontal lobe, except a portion near the convexity of the hemisphere, was destroyed to within 10 mm. of the folds of the island of Reil, and about 8 mm. of the head of the nucleus caudatus. The mass of white substance connecting the inferior and posterior part of the third frontal convolution and the anterior gyri of the island of Reil with the internal capsule, was uninjured.

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* *Archives of Ophthalmology*, vol. ix., p. 185.

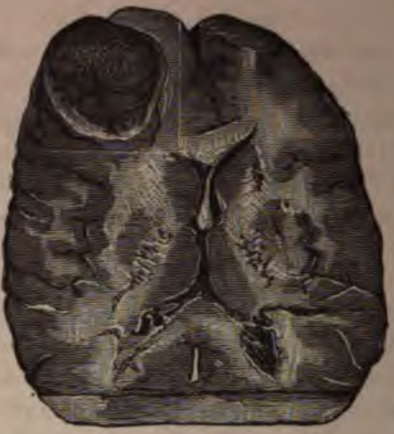

FIG. 11.

Relations of the abscess as shown in a horizontal section of the brain made at the level of Broca's speech-centre. Drawn from a photograph of the specimen. Occipital lobes cut off.

This fact is of capital importance in estimating the bearing of this case upon the current notions of cerebral localization.

The above description of the topography of the lesion, especially its posterior limitation, is made from the surface exposed by the lowest cut made, viz., one passing through the speech-centre of Broca, about 10 mm. above the apparent commencement of the fissure of Sylvius (pia still adherent). Fig. 11 is faithfully drawn from a photograph taken of this section-surface. The rest of the brain was healthy to the naked eye.

This remarkable case seems to me of much importance as a negative contribution to cerebral localization. It is in exact accord with recent experimental data, and with the *post-mortem* findings of the last ten years, that an abscess placed like this one should give rise to no motor symptoms, and should not cause aphasia. It is wholly within what are now called the inexcitable districts of the brain. The only symptoms present were the partial paralysis of the left third nerve (more immediately caused by the orbital abscess?) and signs of intracranial pressure. Yet it is important to note that in spite of the enormous pressure which must have existed, there was no actual neuro-retinitis.

I have elsewhere reported another case of (smaller) abscess in precisely the same location (left frontal lobe) in which no symptoms referable to this lesion were present.*

On the other hand, numerous autopsies are on record in which a smaller lesion (softening, hemorrhage, etc.), placed a centimetre further back in the left frontal lobe, involving the posterior part of the third frontal gyrus or the band of white substance between it and the nucleus caudatus, has given rise to severe symptoms, hemiplegia or aphasia, singly or combined.

In the paper just quoted I have described such cases.

This case has the same negative importance as Case IV.; serving to indicate the anterior limit of motor activities in the hemisphere. It shows that the lower part of the first and second frontal, and the orbital lobule of the frontal lobe, have no direct motor connections with peripheral parts of the body; and, also, that these regions of the brain are non-excitable.

CONCLUSIONS.

The following conclusions may be legitimately drawn from the cases of localized cerebral disease (twelve in number) which I have published in the last four years:

1. The motor area of the cerebral cortex and allied white substance extends anteriorly as far as the lower half of the second and first frontal gyri, and posteriorly as far as the anterior part of the interparietal fissure. This statement is justified by Case VII. of my first paper (lesion of the left frontal lobe), and Cases IV. and V. of the present paper.

2. The region lying between the limits indicated above, the middle regions of the hemisphere, on its convexity and (to a certain extent) on its median surface, including the posterior parts of the first and second, the whole of the third, frontal gyri, the whole of the ascending frontal and ascending parietal gyri, with their terminations in the longitudinal fissure known as the paracentral lobule, with probably the upper parietal lobe—all these cortical parts, with their associated segments or fasciculi

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* See p. 220; also *Transactions of the American Neurological Association*, vol. ii., pp. 122–4, N. Y., 1877.

of white matter, have strong motor functions, being in direct relation with the muscles of the face, tongue, arm, and leg. This general statement is supported by the remaining nine cases in the two essays, in which destructive lesions of this area gave rise to spasm or paralysis on the opposite side of the body.

A further and more elaborate induction is permissible from these nine positive cases:

a. The lower part of the third frontal gyrus is intimately connected with the organs of speech (and the function of language).—Cases I., II., III., and IV. of former essay.

b. The middle parts of the ascending frontal and ascending parietal gyri are directly connected with the arm of the opposite side.—Case V. of first essay, and Case I. of present paper.

c. The upper or posterior part of the ascending frontal and ascending parietal gyri, and the paracentral lobule (also the upper parietal lobule?), are directly connected with the lower and upper extremities of the opposite side, and perhaps more closely with the leg.—Case VI. of first essay, Cases II. and III. of present paper.

I cannot offer any case bearing on the questions of the location of the facial and ocular centres; though I now have under study a living case of exquisite epileptiform facial monospasm, which has been controlled by a strict bromide treatment.

ON THE USE OF A FEEBLY ALKALINE WATER AS A VEHICLE FOR THE ADMINISTRATION OF THE IODIDE AND BROMIDE OF POTASSIUM, ETC.*

ONE hears a great deal in remarks and debates at medical societies and in private consultations of the gastric derangement produced by remedies which are of constant use and of unsurpassed efficacy, viz., the iodide of potassium and the various bromides (more especially the bromides of potassium and sodium). This evil result, or the dread of it, is not infrequently interposed against the free use of these salts in large doses for the relief of serious symptoms.

For example, a patient lies comatose from cerebral syphilis, and when the advice is given to administer .18–.25 potassium iodide every two or four hours, the attending physician very often expresses his fears that great gastric derangement will result, interfering with the digestion of food. I have known the recovery of such a case placed in the greatest jeopardy by such a dread of the local effect of this remedy.

Again, a patient is allowed to have recurring attacks of epilepsy while using small doses of potassium bromide, whereas by giving larger doses the paroxysms might be indefinitely suspended. The larger doses are not given partly from a fear of bromism in general, but also, I am convinced from numerous consultations, because it is believed that the bromides cause gastric catarrh.

I am perfectly ready to admit that the salts in question may and do cause gastro-intestinal disorder, but I have very rarely observed this in my practice during the last three years. Having, as I believe, found the means of administering the iodide of potassium and the various alkaline bromides in a harmless way (as regards the digestive organs), I fancy it may be of some utility to give a detailed account of my plan of administration.

This plan includes the almost equally important conditions:

1. The use of a simple aqueous solution of the salt.

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* Reprinted from the *Archives of Medicine*, vol. vi., No. 1, August, 1881.

2. Its ingestion upon an empty stomach (fifteen or thirty minutes before food).

3. Its very free dilution with an alkaline solution.

I. The importance of employing absolutely simple solutions of certain remedies, especially of the bromic and iodic salts, is being more and more realized by physicians, and the nauseous and, as I believe, indigestible mixtures which were imposed upon the profession by high authorities some twenty years ago, are passing out of use. Certainly, in the case of drugs whose remedial effects are as special and relatively simple as are those of the bromides and iodides, it would seem, *a priori*, that giving them in the shape of an aqueous solution were best. Their efficacy can hardly be increased by the addition of other drugs, and their taste certainly cannot be covered up or neutralized by infusions, syrups, etc. It has been my practice for several years to employ a solution of iodide of potassium made by dissolving equal parts by weight of the salt and of water. Experimenting upon a considerable bulk, it has been found that there is a loss by volume of one-fifth in mixing the salt and water. In other words, a drop of this solution contains about .05. A patient who takes a dose of one hundred drops of this solution does not in reality receive (as is often erroneously stated) 6. of the salt, but only about 5. This difference is of considerable importance in the treatment of cases requiring the maximum doses of iodide. Of this solution I direct that so many drops be given in the dilution to be presently described, about half an hour before meals, or before food.

The bromides I have for some years prescribed upon one general or typical formula, varying the ingredients to suit different cases, but keeping the standard dose the same. This will be at once recognized as of great utility in treating a large number of cases of epilepsy in private and in hospital practice. It is needless to defend the use of a standard formula from the charge of routine practice, because reflection will show that with such a type formula, the doses for each case can be varied infinitely by subdivision and arrangement of quantities of the solution. This general formula is:

℞ Potassii bromidi, - - -	45.
Aquæ, - - - -	200. cc.

A teaspoonful contains 1. of the salt.

Another formula, which I often employ, is:

℞ Ammonii bromidi,	- -	15.
Potassii bromidi,	- -	30.
Aquæ,	- - - -	200. cc.

Of this solution also a teaspoonful contains 1. of the salts.

All of my anti-epileptic solutions are constructed upon this type: one teaspoonful containing 1. of the salts. Perhaps the formulas require some explanations. They are not intended as examples of mathematical accuracy in dosage, such as would avoid an error of .06. They are constructed for practical use in families, and calculated upon the average capacities of teaspoons. These utensils no doubt vary in capacity, but from my own experiments, and from the testimony of others, medical and non-medical witnesses, I have been led to assume that only about seven teaspoonfuls could be obtained from 30. cc. of solution. Each of my standard bromide formulas contains, practically, 49 doses, which, divided into the total quantity of salts, yields a quotient of very nearly 1. The translations into the metric system also need a word of explanation. They are corresponding and logically equivalent translations, and not all literal translations, such as abound in medical books and periodicals—translations absurdly exact, and only serving the purpose of disgusting physicians with the use of the metric system in prescriptions.

Of these various bromide solutions, I direct one or more teaspoonfuls, properly diluted, to be taken upon an empty stomach.

II. The idea of giving the iodides and bromides on an empty stomach is in no wise new, but is in opposition to what I think is the general practice. Influenced by the (delusive) notion that iodides and bromides produce gastric irritation, most practitioners give them after eating, when they probably undergo more rapid decomposition, and interfere with the process of digestion.

Deposited into an empty stomach, which in normal conditions presents a neutral or alkaline reaction, more especially if guarded by an alkaline liquid, it is a practical reality that these salts are very efficacious, and that they cause no gastric irritation. Theoretically it is almost demonstrable that they are absorbed quickly, wholly, and with little if any change. The contact of the solution may act as a solid body or a portion of aliment, and cause an outpouring of acid gastric juice; this is, however, not proven, and if such an event does occur, the acidity thus produced will be antagonized by the alkaline salts of the solution.

III. While serving as resident physician in the New York Hospital in 1865–7, I noted the addition of potassium bicarbonate to prescriptions for potassium iodide by some of the visiting staff. I did this myself in practice afterward, but found objections to the device in that it caused the insertion of one more ingredient in a formula which might already be complex enough, and in that often too much alkaline salt was given. Some five years ago I began directing patients to measure out their dose of bromides or of iodide into a glass, and add a liberal quantity of Vichy water, from one-half to a whole glassful. Gradually I adopted this as a vehicle in all cases, and now testify to the excellent results of this practice from a three years' large experience. When the patient resides in a city or large town, I direct him to procure the artificial Vichy water in siphons, which is now so widely manufactured. Some of these imitation waters are very honestly made nearly like the known composition of the waters of Vichy, and others, the majority, I suppose, are carelessly compounded. At any rate, the siphons contain a solution of bicarbonates of sodium and potassium highly charged with carbonic acid gas, and this is sufficient for our purpose. For patients living where the siphons cannot be procured, or for patients who travel much, I direct the purchase of the effervescent Vichy salts, either of American or foreign manufacture. A teaspoonful of the salts in a glassful of cold water, makes a sparkling glass of Vichy water, in which the medicine can be mixed.

In the case of patients who cannot afford to buy these preparations, I recommend that a good-sized pinch of bicarbonate of sodium be added to a glass of water. The advantages which I claim for this method of giving bromides and iodides in weak alkaline waters surcharged with carbonic acid are two-fold:

First, the supposed irritating effect of the salts upon the gastric mucous membrane is reduced to a minimum, if not absolutely neutralized. This statement is theoretical, but, practically, I am able to state that I almost never observe gastric or gastro-intestinal disorder while giving full or even very large doses of the salts to patients of various ages. I am thus enabled to administer from 5. to 10. of the bromides in the day; and even when bromism occurs, the gastric symptoms are almost *nil.* The iodide of potassium I have thus given in doses varying from small doses to 32. in the day, without indigestion. Occasionally for severe cerebral symptoms, I have caused children to have a dose of 5. three times a day, with only good results.

Second, the taste of the bromides and iodides is considerably masked by the sparkle and sub-acid taste of the effervescent drink. Many patients have thanked me warmly for having substituted a simple solution of bromides (or of iodide of potassium) given in Vichy water for the classical mixtures which they had formerly taken.

I should add that the salicylate of sodium is well taken in this way: a powder of the size required, 1. to 3., is dissolved in a glassful of Vichy water. This covers the disagreeable sweetish taste of the salt, and I believe, favors its complete absorption.

In some simple cases of epilepsy I give only one dose of bromide of potassium in the day, at bed-time or on rising. In such cases I prescribe the medicine as a powder of from 2. to 4. or more, to be taken in a glass of Vichy water.

In some neurasthenic cases, and some cases of oxaluria with insomnia attended by restlessness, I have obtained excellent results from the use of a powder containing (usually) 2. each of salicylate of sodium and bromide of potassium taken in a glassful of Vichy water.

HISTORY OF ATTEMPTS MADE TO CURE THREE CASES OF CHRONIC TRIGEMINAL NEURALGIA.*

It must have appeared to many physicians besides myself that the custom of reporting only successful cases, and of slighting, or altogether omitting, an account of our unsuccessful attempts at cure, was a bad one, and this for several reasons. One of these is that the perusal of such one-sided reports is quite sure to inspire some of our *confrères* with undue confidence in the power of drugs over disease, and to shape their prognosis accordingly.

Among the diseases which most tax our patience and therapeutic skill, there are few more redoubtable than chronic trigeminal neuralgia, or tic douloureux. Excellent as is the reputation of this affection for incurability, yet the published records of this committee embrace several instances of its cure by drugs in patients who had suffered fourteen years or less (*vide New York Medical Journal*, December, 1878, p. 621).

I propose this evening, for the purpose of enabling you to profit by my unsatisfactory experience, to relate briefly the history of three cases of the disease in question which have not been cured.

Case I.—Mr. F. O., aged 45, oyster dealer. History taken when first seen, Dec. 12, 1878. General health has always been excellent. In 1856 had a single malarial chill, followed by two slight attacks of right supra-orbital neuralgia.

Present tic douloureux began in 1857, by a few "sticking" pains near the right infra-orbital foramen: a single pain like the pricking of a needle several times a day. This pain steadily increased in severity and frequency. Came north from Georgia in 1858, and for one year was free from pain. After that time it returned. Two or three times a year afterward he had spontaneous relief for some weeks. In the last two or three years constant suffering. Patient has tried a good many medicines without relief.

Now has a paroxysm of pain every two or three minutes, day and night Eating, drinking, talking, attempts to wash or wipe the skin of the face o right side excite paroxysms of pain.

About three years after commencement of trouble (1857) the pain extende to the whole of the upper maxilla, later to the lower jaw, and recently the whole of the right trigeminus, lingual branch included, has been the seat of pain. There is no regularity or periodicity in time of appearance of the pain, or in its degree of intensity. The patient never has common headache or dizziness. In 1857–58 one tooth was pulled from the right upper jaw, and another in 1867 ; pain aggravated each time.

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* Reprinted from the *Archives of Medicine*, vol. vi., No. 1, August, 1881.

Denies injury to face and syphilis.

Examination.—Patient is a large and powerful man, of healthy aspect, with a facies indicative of suffering. Every few minutes he has an epileptiform (*i.e.*, sudden) onset of pain in right side of face and head to vertex; pain sharp and cutting; paroxysm lasts a few seconds, and during it the face flushes. The cutaneous sensibility of the affected region is normal to simple touch and to æsthesiometer test. Hearing of right ear O, drum thick and whitish. Hearing of left ear 30–38 cm. (watch). The corneæ are normal; right pupil is a trifle smaller than the left in intervals between pains. The teeth on right upper and lower jaws are covered with an extraordinary layer of "tartar," and some are loosened. Patient has not dared cleanse teeth on that side for years. There are no tender points upon the face or in the mouth. Teeth on the left side are fairly clean.

Was ordered solution of Duquesnel's crystallized aconitia, in doses of .001, and this was given in increasing doses, with no relief. On December 18th following note recurs: Aconitia must be deemed a failure. Has taken .006 in 24 hours. Constant great effects on sensory nerves, coldness and tingling. Has pains almost every two minutes. Fowler's solution ordered in increasing doses after meals. December 30th. Has increased arsenic solution to sixteen drops after each meal; nausea; no relief to pain.

Ext. gelsemii fluid ordered December 30th, five drops before each meal, and at bedtime. January 14 (1879), full effects of gelsemium obtained from doses of fourteen and fifteen drops four times a day. No relief to pain.

Sol. phosphori, Thompson (teaspoonful containing .003 of phosphorus), tried in doses of 4 cc. an hour before each meal for several days; no effect.

Injections of chloroform in cheek used on January 20th, 21st, and 22d. Injections made through mucous membrane, toward right infra-orbital nerve. .30 on 20th, .60 on 21st, with no relief; slight swelling and burning pain. Attempt to inject 1. cc. on 22d resulted in asphyxia, and apparent death, previously reported to the committee.

Mixed treatment, iodide of mercury and saturated solution of iodide of potassium, ordered on January 23d. On February 11th slight effect on gums is noted; takes about forty drops of sol. sat. KI. three times a day; no relief.

Galvanism, stabile, strong current (25 cells); cathode on tender points from 7 to 15 minutes. Patient thinks pain is aggravated by the current.

Ammonio-sulphate of copper ordered, .08, with ext. cannabis ind., .03, before each meal since February 11th; stopped on 15th; no relief.

Operation.—Resection of right infra-orbital nerve performed in February. Nerve removed outside and inside orbit. Healed by primary union.

March 9th.—Face perfectly healed; only part that is absolutely anæsthetic to faradic current by brush is a spot about 2 cent. square under right eye. Has partial sensibility to brush, and pricking in rest of cheek, in ala nasi, and upper lip, and inner aspect of cheek and mouth. To-day less pain, but he suffered very much on 6th, 7th, and yesterday. A paroxysm seen in office seems less severe than those before operation. Ordered quinia sulph., .25, morphia sulph., .02, three times a day.

March 14.—Much better. Few attacks in supra-maxillary region. Talking and chewing can be done without agony. Has had several severe attacks of pain in infra-maxillary region, and in outer part of orbit; not in supra-orbital district. Has had good nights. Continue quinia and morphia.

March 19.—Is fifty per cent. better than before operation (patient's own estimate).

March 28th.—No "neuralgic" pain in right upper jaw and lip, but the lower jaw and lip are seat of severe neuralgic pains, not as severe as formerly. Ordered pil. quiniæ et morphiæ et belladonnæ twice a day. Ordered fluid extract of aconite, one drop *t. i. d.*

March 31st.—No neuralgic pain in upper jaw; severe in lower jaw. Continue aconite.

April 14th.—Considers his condition improved at least 50 per cent. Takes .25–.30 tincture aconite, with slight physiological effects. Sleeps soundly. No severe paroxysms in two weeks.

During May more pain; severe paroxysms in anæsthetic district. Fowler's solution, aconite, morphia again tried in vain.

Was not seen again until December 17, 1880. Was free from extreme suffering for several months. In last few months almost constant severe pain.

I have since tried aconitia and gelsemium to physiological effects, without relief.

Dr. Weir is planning to remove Meckel's ganglion.*

CASE II.—Mr. H. S., janitor, aged 29 years. History of case taken October 2, 1878 (*vide* a partial report on the case in New York *Medical Record*, January 4, 1879, vol. xv., p. 7).

Previous to the development of the present affection, he had been subject to occasional dull headaches. Ten years ago (1868) he suddenly experienced a very severe sharp pain all through his head, "as if devils were at work there," lasting half an hour. There was no dizziness or faintness, or nausea, or impairment of sight, or paralysis. For a period of six months he remained free from pain, and, indeed, was perfectly well. After that time, nearly ten years ago, a "dull, stupid pain" began over the right eye, extending from the supra-orbital notch inward to the nose, and down the side of the nose to the ala nasi. This pain was paroxysmal, and worse in the daytime. Later the pain extended to the eyeball, and was exceedingly severe, the paroxysms recurring from ten to twelve times a day. In the course of two or three years the pain made its appearance in the right temple—worse at night.

In the last few years most of the pain has been on the top of the head, above the temple, and in front of the ear to the bregma. There has lately been only an occasional pain in the side of the nose, and not much pain in the temple proper. During the past summer, and since, there has been some occipital pain on both sides—more on the right. In the last year there has also been pain in both the upper and lower jaws, in the upper lip near the median line; none in the tongue (on right side). In the last four years vision has been dim, and glasses have not remedied the defect. Five years ago had temporary diplopia, but this was while taking some unknown medicine. At various times during this long illness has had "dizzy spells," with varying frequency; few in the last months. Has had no other symptoms of a neuralgic nature. Memory is impaired and virility quite lost. Had severe dyspepsia and vomiting three years ago, and has been costive during the whole period of the disease.

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* The operation was subsequently done with relief.—[R. W. A.]

Examination.—The various painful regions are hyperalgesic, but not numb, and the tactile sensibility is perfectly preserved on both sides. There is no facial paralysis; the right pupil is positively small, the left normal. After dilatation by atropine opthalmoscope reveals no lesion in the fundus. Hearing, smell and taste are normal. Cornea clear. The urine (frequently examined by other physicians and found normal) is now free from albumen. Marked anæmia is exhibited by the skin and mucous membranes; has always been pale; denies syphilis.

The pains, which occur frequently in my office, are the most terrible which I have ever witnessed; the patient fairly writhing in his chair, or even falling to the floor (not unconscious) in his agony. During the attack the right eye is much injected, and tears flow freely from it, while the left eye remains dry.

The patient states that no medicine has ever relieved him, and that he has tried a great many.

The treatment in this case, though prolonged until now, February, 1881, has been relatively simple.

Duquesnel's aconitia in doses of .0006. Solutions by Neergaard at first, later in the shape of Schieffelin's granules, given from two to four times a day. Full physiological effects were easily obtained, and were kept up for many months. Numbness and a remarkable cold chilly condition were the signs. At times the subjective cold was so great that he would come to my office shivering in an overcoat.

In this case, as in Case III., increased susceptibility to the action of the drug was observed as time went by. In the last few months one dose of .0006 produces effects which last from six to nine hours.

Besides aconitia, iron and Fowler's solution in moderate doses have been administered frequently. Has had several attacks of subacute rheumatism rapidly cured by sodium salicylate.

On the whole the result obtained is very gratifying—it is a relative cure.

Patient a few weeks after beginning of treatment experienced no excruciating paroxysms and gradually resumed his occupation as janitor. In last few months seldom loses half a day. Has kept a record of attacks, classifying them into severe and mild; has had very few severe ones in each month, and has registered many days without any pain.

There has occurred a curious shifting of pain. It was formerly more intense in fronto-temporal region, it is now developed mostly near the parietal eminence.

The patient's general condition has greatly improved; he still has a peculiarly white skin, but his lips, etc., are fairly well colored.

The change in *moral* is most remarkable; is now cheerful, and enjoys both his work and his family pleasures; whereas about a year ago he looked upon life as a burden.

CASE III. Mr. W. L. P., clerk, aged 54 years, seen September 22d, 1880.

Had always enjoyed good health.

In 1876 there appeared a pain in front of the right temporo-maxillary articulation; a deep pain. At first the pain was occasional, excited by washing face. Pain has steadily increased in frequency and severity, until now paroxysms occur almost every moment. The pain is rather worse in afternoon and night, not typically nocturnal. In about a year after beginning, the pain extended to infra-maxillary and infra-orbital nerves (never appearing at mental foramen). It extends into the gums in right upper and lower jaws, and "strikes" in the lower jaw at a point a little posterior to the angle of the mouth. No pain above zygoma and orbit. Saliva flows in the paroxysms. All movements of jaw cause more pain. Weather is without influence.

No malarial fever since his 16th year. Never had syphilis. Used much tobacco until recently. Temperate. Has had seven teeth pulled from the right upper jaw without relief.

Examination.—No tender point, except at the mental foramen, where there is no pain. No evident anæsthesia. Some atrophy of fatty tissues of face on the right side. Opening mouth causes a paroxysm. Hair on face kept stubby, and is worn on cheek by constant friction of hand and fingers during paroxysms. Attacks last from one to one and a half minutes. General health good.

The treatment was began September 22d, by giving Duquesnel's crystallized aconitia, in the shape of tablets made by Caswell & Hazard, .0003, every two hours.

25th.—No strong aconitia effect. Sleeps without chloral. Ordered, .0003 every hour. To-morrow .0006 every two or three hours.

27th.—Great relief; did not feel aconitia much, .0006 every two hours till 5 P.M., when he was quite numb, and sight was dim.

29th.—Marked improvement; pain only in zygomatic region. From the 29th to October 1st, included, sol. phosphori Thompson was used, teaspoonful three times a day. Pain made worse. Again given .0006 aconitia.

Oct. 4th.—Severe pain; no aconitia for one day. Takes sol. Fowler., eight drops after each meal, increasing. Ordered continue Fowler, and take ext. gelsemii fld., five drops every two hours. Continue and increase the Fowler's solution.

10th.—Ext. gelsemii fld. is being used, but no aconitia. Takes eight drops of gelsemium every three hours with moderate effect; double vision at times; lids heavy. Very little severe pain; has lost habit of rubbing cheek in paroxysms; good nights.

20th.—Has reached a maximum dose of sixteen drops Fowler's after each meal. Gelsemium as above. The gelsemium is stopped; Fowler's continued, and aconitia, .0006 every two hours, ordered.

23d.—Very little pain in last forty-eight hours; feels the aconitia; attacks slight; pain nearly localized near right temporo-maxillary articulation; can eat and talk with little pain.

Iodide of potassium, saturated solution, in doses of twenty drops before each meal in much water, increased by five drops daily, was begun on 28th. Fowler's abandoned. Aconitia, .0006 *p. r. n.*

Nov. 3d.—Coryza and hoarseness, neuralgia slight. Takes forty drops *t. i. d.* Stop. Cautery on focus of pain in front of ear tried on 5th. Pain aggravated.

Ammonio-sulphate of copper was tried during November, December, and January (1881), given in pills, dose increased from .05 *t. i. d.* after meals, to .20 after each meal and at bedtime. Aconitia .0006 was used *p. r. n.* by patient all the time.

Pain very variable; a few days almost without pain; other days much pain, often under influence of storm or rain. At one time copper *before* meals produced griping and watery stools; no ill effect when administered after food.

Dec. 11th.—The note is made that patient has become much more sensitive to aconitia; is affected in ten minutes by one tablet, whereas formerly it required an hour or more to obtain any prickling.

The whole of January, 1881, was very comfortable.

1st.—No severe paroxysms. Was in Canada part of the time. Now can use only one or two (.0006) tablets of aconitia a day—formerly could take one every two or three hours.

Feb. 19th.—In last month gelsemium and aconitia. Much more pain in last fortnight, though not as much as before treatment. Pain is severe in spite of full effects of gelsemium, five drops every two hours.

A fair summing up of these attempts at relief of incurable conditions is, it seems to me, that aconitia is the chief agent to be relied on for the alleviation of the pain of chronic trigeminal neuralgia, and for its cure. Of course, malarious and syphilitic neuralgias are excluded from this statement; in them we have special indications.

Gelsemium and arsenic have both seemed to exert a secondary beneficial influence.

Galvanism, the actual cautery, injections of chloroform, were useless. Morphia and chloral afforded mere temporary relief.

IMPORTANCE OF THE EARLY RECOGNITION OF EPILEPSY.*

THE paper which I have the honor to submit to the Society is one which treats of an eminently practical subject, handled, I trust, in a practical manner. It is upon the importance of the early diagnosis of epilepsy in its principal forms. This essay is also a logical correlative of the one which I presented to the Medical Society of the State of New York in February of this year, upon the early diagnosis of some organic diseases of the nervous system.†

My attention has for several years been attracted by the fact that most cases of epilepsy were allowed to go on for months or years, perhaps passing the stage of curability, without a correct diagnosis and proper treatment. The mistakes of this sort which come under the observation of specialists are very numerous, and many of them have been committed by leading members of our profession. I shall present a number of histories of cases illustrating these errors of diagnosis, and I would earnestly request my readers to bear in mind that I cite these mistakes only for the purpose of instruction, and not at all with the idea of fault finding, or of exalting my own diagnostic acumen. Several of the physicians referred to anonymously in the following pages are gentlemen who are really eminent as teachers and practitioners, and at whose feet I would be glad to sit. Their errors were not due to carelessness or ignorance, but to a too ready acceptance of medical laws which pass current, yet are wrong.

The subject in hand is really a very complicated one, and I cannot pretend, in a paper whose length is limited, to enter into full details concerning the diagnosis of all the forms of epilepsy, and of the various symptom groups which may be mistaken for it. To do this would involve a prolonged and minute discussion of many mooted points.

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* A paper read before the Connecticut State Medical Society. Reprinted from the *Medical Record*, Aug. 6 and 13, 1881, vol. xx.

† See the *Medical Record*, 1881, vol. xix., p. 225.

All I can do is to show that in most cases even the first attacks of epilepsy, of grand-mal and of petit-mal, can be recognized as epileptic.

I shall first relate cases in which the grand-mal, or regular spasmodic attacks, were allowed to go on without proper treatment for want of a correct diagnosis, and offer some comments on each case.

CASE I.—Female child, aged eleven years, seen May, 1881. Was always a bad sleeper, of a restless disposition, and irritable. Was easily made to turn pale under excitement. Nights disturbed by talking, crying, and even by nightmare. Five years ago the family physician regulated child's diet and instituted some simple treatment; since which attacks of pallor have been infrequent—very rare during the past winter. Never had chorea. Indigestion has been a prominent feature in the child's life; she was fond of sweets. The urine was often found laden with oxalate of lime, and once, last summer, a trace of albumen was discovered.

As regards any tendency to epilepsy, it appears that the child's mother is very nervous, and that one of her brothers was epileptic from childhood, in consequence of a fall (?).

In June of last year the patient travelled about somewhat, alone with her father, without the supervision of mother and nurse. Was probably a great deal exposed to the sun. On or about June 20th, in the early evening, while out on the grass, fell in a severe convulsion. Seemed in poor health afterward, and the family physician, considering the attack as caused by indigestion, or at any rate as symptomatic, prescribed a strict diet and an occasional dose of calomel.

On September 29th, in the cars, returning home from the country, was excited and overheated. After arriving at the house had her second attack, characterized by loss of consciousness, universal spasm, frothing at the mouth. Did not bite tongue, and was not inclined to sleep after fit. Traces of albumen in urine. Treatment was still directed to the disordered state of the digestive apparatus as a cause of the epileptiform attacks.

A week later, October 6th, after some excitement, in presence of her mother, suddenly fell in a convulsion, lighter than the previous ones.

After this a moderate bromide treatment was added to the management of the case, and no attack occurred until April 25, 1881.

The bromides, though given judiciously and by a very skilful hand, produced irritability and other disagreeable symptoms, which led the child's mother to cease giving them some time in January.

An attack on April 25th was characteristic, but slight, and followed by sleep. After it urine found laden with oxalate of lime.

May 2d, while the child was being prepared for bed, she felt some warning sensation (not a definite one); rushed, nearly undressed, to her mother, stood speechless before her, and was slightly convulsed in the throat.

A sixth and last attack occurred in the evening of May 8th. Sat in a spasm, foamed at the mouth; was rigid one instant, and then had clonic spasms of extremities; face not convulsed (?); pupils not observed.

There have been no "dizzy spells" or petit-mal.

It is needless to add that I advised a resumption of the bromide treatment in this typical case of epilepsy, with special precautions against severe bromic symptoms.

This case is peculiarly instructive, because the physician who treated it as one of lithæmia and oxaluria, with symptomatic convulsions, is an unusually intelligent practitioner and a gentleman of high standing in the profession. His judgment was warped by the currently accepted notions of the frequency of eclampsia.

CASE II.—Mrs. C. S., aged thirty-four years, seen March, 1879. Former health good. During 1876–77 she had had much malarial fever, irregular chills, and attacks of fever.

In April, 1878, after having been confined to the house by an attack of fever, she had a first convulsion. This occurred in sleep, after dinner, at about 2 o'clock P.M. It was a full convulsive seizure, in which she bit her tongue, fell heavily from the lounge on which she was lying, and hurt her face.

A second attack recurred on the night of August 2d. She groaned, was convulsed, frothed at the mouth, and bit her tongue.

A third attack of grand-mal occurred in the night of January 1–2, 1879. This was less severe, but she bit her tongue.

Has had few attacks of petit-mal, consisting in momentary confusion.

The significance of the first attack was ignored in this case, and a bromide treatment was not begun until after the second seizure. Since then has had bromide irregularly, at times too little, sometimes none at all, and occasionally too much.

Etiology obscure. Owing to patient's age and the absence of any inherited tendency, I inquired particularly as to symptoms of syphilis, with negative result. Frequent examinations of the urine have shown no sign of renal disease. As regards syphilis, the subsequent course of events, improvement without mercury or iodide of potassium, has justified my conclusion at the time of my examination.

It is sufficient to state, with respect to treatment, that Mrs. S. was put on a careful course of chloral and potassium bromide, which last year was changed to ammonium and potassium bromides. She has never required very much of the anti-epileptic medicines, and at times has had various tonics.

To the present time, May, 1881, a period of twenty-nine months, she has had no attack of either kind, and her general health is excellent. Since the beginning of the year I have made a small reduction in the amount of bromide, and intend to make a further reduction every three months. Her present dose of the mixture of ammonium and potassium bromides is .50 on rising and 2.5 at bed-time.

If, after exclusion of uræmia and syphilis, the first attack had been diagnosticated as epileptic, and treatment instituted, the probability of cure would have been greatly enhanced. The next case is an illustration of this statement.

CASE III.—Miss F. O., aged sixteen years, seen May, 1878. A well-developed, healthy girl, menstruating since three years, with little pain. Mother neurasthenic; one brother had an exquisite attack of articular neuromimesis (both ankles). Patient never hysterical.

Yesterday, May —, menses were flowing, when, in order to be able to go with comfort to a dancing-school *soirée*, she used a cold foot-bath and checked the flow. She danced a good deal in the course of the evening, and then took supper. To-day arose late, and seemed languid. About seven P.M. had a severe fit; gave a cry, lost consciousness, fell heavily; body was rigid and pupils wide open, then had clonic spasm, frothed at the mouth, and bit her tongue; was stupid and sleepy after attack, whose actual duration was not timed. It was witnessed by a very intelligent young gentleman, who gave me the above particulars. I saw the patient at eight o'clock—one hour after the fit. She was conscious, complained of headache, of soreness in body generally, and of her bitten tongue. The pulse was rapid, the axillary temperature was over 37.8° C.; the patient's face and neck were covered with numerous minute petechiæ resembling flea-bites; menses had returned.

The seizure was typically epileptic. I anticipated a return of attacks only at the menstrual periods, and consequently instituted a rather peculiar treatment, which was carried out with unusual faithfulness. Bromide of potassium, 1.50, was to be taken night and morning for ten days, including the menstrual period; beginning three or four days before it; and the patient was to be kept in bed or on the lounge for two or three days at the beginning of the flow.

A few days ago (May 14, 1881) I had a note from the patient's mother, stating that her daughter had never had a return of spasm (or any other epileptic manifestation), and that she was still keeping quiet for two days in the menstrual week, and taking bromide of potassium.

This makes an interval of more than three years, and I must say that I consider a return of attacks exceedingly unlikely. Still I have recommended continuing the periodical treatment for six or eight months longer.

CASE IV.—Mr. C. D——, aged twenty-one, seen February, 1877. Patient is a large and well-developed young man, something of an athlete. Former health good, but hygiene bad; used wine and tobacco from twelve to eighteen years, and probably committed sexual excesses. No epilepsy in family; mother subject to migraine; patient not. No dizzy spells. Head not injured.

First attack occurred when he seemed in good health, on Christmas day, 1873. Was sliding down hill, when he lost consciousness and fell off the sled, remained stiff for a few minutes, and was sick at his stomach. From his knowledge of the circumstances and from what he was told, the patient is positive that the fall from the sled was not accidental, but that he first "fainted." In two or three days was well.

Remained perfectly well for ten months, and had a second attack in October, 1874. Was sitting chatting with friends; lost consciousness, became sick at the stomach ; did not bite his tongue. Had muscular soreness the next day. In the summer of 1875, after rowing on Lake George, had a third attack, without aura ; fell off a dock into the water. Fourth attack in May, 1876, preceded by an undefinable preliminary sensation ; attack was again accompanied or followed by vomiting. Fifth attack in October, 1876. This was treated by Dr. X., as stated in the letter which is appended. Another attack occurred toward the close of 1876, and the seventh (last) seizure was on February 9th of the present year (1877), without warning or vomiting. In the other attacks the warning sensation was quite prolonged ; on one occasion was able to walk nearly a quarter of a mile before falling.

He has had no petit-mal, and general health has been unimpaired. Has done well at college.

The following letter was sent to me with the patient, and it well illustrates the erroneous notions which prevail with respect to the significance of a single epileptic fit, or of fits returning at long intervals.

NEW YORK, *February* 11, 1877.

"———:

". . . . This will be handed you by Mr. D——, who has been under my care for some time with epilepsy. At first I attributed his attacks to gastro-intestinal causes, and rectified all bad habits of life and regimen. The disease recurred, and then I put him upon a diet exclusively vegetable and interdicted stimulants. He will tell you how he has fared upon this plan. . . ."

It appears that Mr. D—— was first prescribed for by Dr. X., who is a very eminent practitioner and teacher in New York, after the fourth or fifth attack, in 1876, when the disease had been going on two years and more. No bromide of potassium or sodium had been given until within a very short time previous to the consultation.

Though foreign to my present purpose, I might add that a bromide treatment, consisting in giving only a night dose of from 4. to 5. of bromide of potassium, has greatly improved the patient.

After the consultation some attacks returned, and I find the following record, July 16, 1878: Mr. D—— returned from Germany a few days ago. He had had no attack since the beginning of August, 1877. Once in Europe had a slight threat of attack, without loss of consciousness. Has led a regular, quiet life, and has taken 4. of potassium bromide every night without omission. General health excellent.

August 4, 1879.—No attack since threat in Germany, twenty months ago. Takes 4. nocte ; ordered reduce to 3.5.

October 19th.—Slight attack after an interval of twenty-eight months; fell and bit his tongue. Ordered 4. at bed-time.

December 6th.—No attack.

September, 1880.—In August, under excitement, felt faint, but this attack was not sudden, and he preserved his consciousness. It is now nearly one year since last attack. Takes only 2.75 bromide of potassium at night. He reduced without advice.

October 14th.—Slowly developed attack, without local aura; felt confused before losing consciousness; had spasm, but did not bite tongue. Ordered 4.

January 5, 1881.—No attack. Through erroneous weighing of bromide, has taken only 3. every night. Ordered 4.

March 23d.—No attack.

Summary.—Since August, 1877, only two epileptic attacks, and one "threat." This is a period of now (June, 1881) nearly four years.

Has finished the study of law, and is in good physical and mental health.

CASE V.—Ira K., aged eight years, seen February, 1877. Was a healthy baby; no convulsions while teething. When two years old fell down a long flight of stairs without apparent injury. Remained well. When four years of age, fell from a horse, cutting the scalp in the occipital region; no loss of consciousness or vomiting.

In six or seven months after this injury, about three and a half years ago, had a first (?) nocturnal epileptic attack. Until lately has had chiefly nocturnal spasms. At first had a few diurnal seizures, and again lately.

Has had much petit-mal, increasing in frequency—of late almost daily. This consists in staring, loss of consciousness, a "hum" or "hem" noise, and sometimes slight jerking of the arms and throwing back of body.

In the last few weeks child has been less bright, and has exhibited a thick articulation.

During the long periods of time the child was treated "for worms" and for "disorder of the stomach."

CASE VI.—Mary C——, aged sixteen years, seen October, 1879. Born healthy and remained well until sixth year, when, after indulgence in green fruit, she had an attack of very severe convulsions lasting two hours; did not bite her tongue, and there was no consequent paralysis.

This was succeeded by numerous "fainting turns," as the mother calls them. In these the child was unconscious, pale, still, with eyes open and staring. This was petit-mal; the next attack of grand-mal occurred in two years, and afterward the convulsive seizures became frequent, from one or two in one day to one in two or three weeks.

The child had an irregular bromide treatment.

Since has had three types of attack: petit-mal (rarely now), grand-mal, and mixed attacks. One of the last kind was witnessed in my office, and is thus described in the case-book: "Makes complaint of aura, asks for amyl, dilatation of the pupils, pale face, general spasm of semi-tonic kind, muttering, raising of clothes, picking or grasping at chair, incoherent remarks, makes some swallowing movements, does not bite tongue, or froth; return to consciousness."

The aura referred to is almost always felt; it consists in a sensation just above the umbilicus, not ascending, but feeling as of a "soft whirling" or "trembling" sensation in the abdomen; no nausea.

I mention this case because of the apparent etiology. It may have been looked upon as a case of convulsion and vertigo from gastric irritations, and treated accordingly. It certainly appears that a serious bromide treatment was not given during the first two or three years of the disease.

Case VII.—Clara C——, aged five years, seen April, 1880. In February 1879, had a first attack of convulsions, on both sides of the body.

In four weeks experienced a second bilateral attack. In April there recurred an attack in which the spasm was wholly limited to the left side of the body, followed by a number of others, all within a period of twelve hours; no consequent paralysis. In the month of May passed through another *status epilepticus*, in which some of the spasms were on the left side, others bilateral. The bromide of potassium was then steadily given until June, when the mother suspended its use. Had no treatment and remained free from attacks until February, 1880, when a *status epilepticus* of forty-eight hours' duration occurred; most of the spasms were bilateral, and a few involved only the left arm and leg; never bit her tongue. Early in March several attacks in a group. Paralysis has never been observed after attacks, but the child is cross and has headache after them. Most of the attacks have been nocturnal.

Recently one dizzy spell.

After this consultation, a stricter bromide treatment was attempted, but never faithfully carried out by the mother.

Status epilepticus occurred in June and September, 1880; many attacks limited to left side. After June attacks, she was almost maniacal for one month.

From January 21st to February, 1881 (when last seen), many seizures, most of them of mixed type, some like petit-mal; calling out, with slight shaking of both arms, staring, and pallor of face. Very unmanageable; semi-maniacal at times in last few weeks.

Family neurotic; maternal grandfather subject to violent neuralgias about the head (specific ?); mother of child had convulsions from eighth year, for how long a time and of what kind it is impossible to learn.

Careful examinations of the child on two occasions, nearly one year apart, gave no objective symptoms indicating the existence of what one would naturally suspect, viz., a localized lesion (tumor ?) in the right hemisphere of the brain.

For many months the physician in charge of the child, and the consulting physician, a man of great eminence, ignored the truly epileptic nature of the child's attacks, asserted their curability, and treated the child carefully for worms and for disorder of the digestive organs.

Case VIII.—Jas. W——, aged twenty-one years, seen October, 1878. Health has been good; denies masturbation, sexual excess, and syphilis. Married fifteen months ago, and has one healthy child.

First epileptic attack occurred three years ago, and the second after an interval of eighteen months. Since the second attack, has had seizures with increasing frequency—thirteen in the last twelve months. Last seizure occurred yesterday. The attacks have all been nocturnal, occurring at from one to five o'clock A.M., and characterized by severe spasm, biting of the tongue, and passing urine in the bed, and followed by heavy sleep. The next morning has headache.

Did not have treatment until after attacks became frequent.

This case is interesting, as showing the real significance of a first epileptiform seizure in a non-syphilitic and non-uræmic adult. The patient had epilepsy just as much after the first attack as he did when the seizures recurred every two or three weeks, and the proper time for successful treatment would have been after the first attack.

CASE IX.—Mary L——, twenty-two years, seen October, 1880. When only three weeks old had a series of convulsions in the course of one week, followed by cyanosis. Afterward was subject to "screaming spells," in which she threw her body forcibly backward.

From fourth to eighth year no attacks of any kind. When eight or nine years of age had attacks of unconsciousness, in which the eyes rolled up, the appearance was statue-like, with a cataleptic state of the limbs. These attacks have occurred daily since; on some days she has had as many as ten or twenty seizures.

Menstruation occurred at thirteen years, but the attacks continued unchanged. Went to school at usual age, but study was abandoned in twelfth year, nominally because of "indigestion," but in reality because patient's mind was feeble; she was to a degree imbecile.

In the last fourteen months has had five attacks of grand-mal; the first in August, 1879, the last one about two weeks ago. In these severe attacks she did not bite her tongue. Has had fewer attacks of petit-mal since these convulsions.

Patient states that she has no aura; as to frequency of petit-mal, she thinks she may have had as many as one hundred " spells " in one day.

The existence of neuroses in the family is denied. The cause of the second series of epileptic phenomena (from eighth year) appears to have been masturbation, which was practiced from the sixth or seventh year until some time after attacks set in. Positively denies self-abuse in the last few years.

This young woman's father was a physician, recently deceased, but the epileptic nature of the disease was not recognized until the convulsive attacks of 1879–80.

CASE X.—Mrs. C. A. R., aged twenty-eight years, seen December, 1878. Was a robust, healthy girl; menstruated in her thirteenth year. In the same year had a very severe attack of typhoid fever, followed by great debility of body and mind. Sexual feeling, which had already been experienced by the patient, disappeared and has never returned. Menses continued nearly regular. At an uncertain time (not long) after the fever, began to have petit-mal of the faintest kind; a mere momentary blurring or loss of consciousness, at frequent, but irregular intervals.

Married at eighteen, and has borne children. Petit-mal has continued occasionally. At about twenty-one had a first convulsion one morning after rising; she frothed at the mouth and bit her tongue. In about two months had a second equally severe attack, and a third one thirteen months later. Then was given bromide and valerian, but irregularly. Four years passed without any convulsions, but she continued having petit-mal at intervals of a few days to three weeks in length.

In 1876 was in Europe travelling, not eating much and using stimulants; had an attack in the summer (grand-mal), and three since. The last one occurred six weeks ago, in the night. Petit-mal occasionally.

In reality this patient was epileptic some twelve years without having a proper diagnosis and treatment.

CASE XI.—Lizzie B., aged twenty-seven years; seen July, 1880. Since the age of eleven or twelve years has had peculiar attacks, consisting of a sensation of something starting in the epigastrium and rolling up to the throat, lasting only a few seconds, not accompanied by tears or other emotional disturbance. Thinks that her consciousness is not lost; calls for hartshorn. At first these attacks occurred once in three or six months; in the last year has had them every two or three weeks. The true nature of these attacks was ignored, though patient was under the constant supervision of a good physician.

Menstruation established at fourteen years (long after first petit-mal); irregular and with pain.

In March, 1880, patient had a regular convulsion, and a slighter one a few days ago. These attacks are described by patient's sister; she herself thinks that they were "long faints." [This shows how much reliance is to be placed on her other statement that she preserves her consciousness in the slighter seizures.]

It was only after the spasm in March that a bromide treatment was instituted. Patient went home in October.

Since January, 1881, quite a number of attacks of grand-mal.

CASE XII.—Dr. ——'s son, aged twenty-two years, November, 1880. Patient not seen; statement made by the father.

From twelfth to sixteenth year had occasional "frightened spells" or "faint spells." No details.

In sixteenth year first recognized epileptic attacks; usually nocturnal; grand-mal at intervals of one or two months. Four attacks in the last four months. In attack spasm begins on the left side of the body, and is most severe on that side; the tongue is bitten, and there is frothing at the mouth.

In 1878–79 had no grand-mal (interval of nearly two years).

At age of six or seven years fell on the ice, striking on his forehead; lost consciousness and vomited.

Masturbation begun in eighth year, probably before petit-mal, which it appears began before the twelfth year, as stated in commencement of the history.

The father, though a practicing physician, paid no special attention to the petit-mal, and attributed the first convulsions to late suppers and gastric irritation in general.

The foregoing cases indicate that the error usually committed in judging of the true nature of first epileptic seizures is in considering them to be sympathetic convulsions, due to remediable causes—in other words, eclamptic attacks.

This capital diagnostic error is founded upon two erroneous conceptions, in my opinion:

1. A physiological misconception. In the first two years of life there is great convulsibility; the spinal axis is excessively irritable, and many causes, local, diathetic, and thermal, may produce convulsions. Thus, pneumonia, exanthemata, infantile spinal paralysis, intestinal worms, gastric irritation, gingivitis, sexual irritation, etc., may cause convulsive attacks, which are usually called eclamptic. If the cause be removed, such attacks do not recur. In the third and fourth years of life, more or less rapidly according to the constitution of the child, this mobile state of the spinal axis diminishes, the inhibitory cerebral influence is more and more shown, and the tendency to reflex spasmodic manifestations almost disappears.

The misconception lies in admitting, beyond the truly infantile age (three to four years), a liability to symptomatic or eclamptic convulsions.

There are exceptions of course—some few children, and even adults (especially females), show convulsibility; but I believe that it may be stated, as a law most useful in estimating the significance of a first fit, that after the third or fourth year eclamptic attacks (except from uræmia) are excessively rare. A first rule for the study of convulsions then is, that convulsibility diminishes rapidly after the third year.

2. An etiological misconception, consisting in over-estimating the exciting powers of local, internal, and peripheral causes. The doctrine of reflex neuroses, reflex neuralgia, reflex spasms, reflex paralyses, and of reflex psychoses, has fallen from the very high standing it acquired, mainly under the influence of Brown-Séquard, some fifteen or twenty years ago. Reflex diseases of all kinds are now rarely reported by reliable observers, and more especially is this true of paralysis. That there are reflex nervous diseases I recognize, but I claim that they are not by any means as common as is usually believed.

More especially would I maintain this with respect to convulsions occurring after the third year of life. Cases of convulsions, or epilepsy, in individuals above three years of age, due to cuts, blows, worms, adherent prepuce, etc., abound in older medical writings, books, and journals; but in the last ten years physicians have become much more guarded, and such cases, when reported, are considered very interesting because of their rarity. Leaving out injuries about the head, I am not sure that I have met with such a case.

I would suggest, as a second safe rule in studying first convulsions, that after the third year of life, local irritations, internal or external, are not likely to cause convulsions without the pre-existence of a morbid state of the nervous centres, inherited or acquired.

The terms eclamptic and epileptic, as applied to convulsions accompanied by loss of consciousness, have been the source of great confusion. The words are often used as if they designated different symptom groups, whereas, in reality, as sanctioned by observations and by the best authorities in our art, they mean the same symptom group, occurring under different conditions.

I might support this statement by numerous citations, and by a minute description of a variety of attacks of each kind; but my time is short, and I will content myself with giving the opinion of a few authorities.

In the first place, Professor Trousseau says: *

"I have often known epilepsy and eclampsia to be confounded one with another, and I have also said that this confusion is almost inevitable, because, if we study only the convulsive manifestations of these two affections, they are indistinguishable.

"If you observe a woman attacked with eclampsia in the eighth or ninth month of pregnancy, or during confinement, or a child who is convulsed, either at the beginning of an eruptive fever or during dentition, however you may be on your guard—however careful you may be in your observations—you cannot make out any [symptomatic] differences between these attacks and the convulsive form of morbus caducus."

Dr. Day, in the recent edition of his excellent work on diseases of children,† quotes with approval Trousseau's statement as to the similarity of eclamptic and epileptic attacks—the latter being the former repeated in a series—and adds, when speaking of eclamptic attacks:

"In many respects they (the convulsions) resemble epilepsy, from which, indeed, they cannot invariably be distinguished."

An encyclopedic treatise on diseases of children is being issued

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* Clinique Médicale de l'Hôtel-Dieu de Paris. Seconde édition, vol. iii., p. 88 Paris, 1865.

† The Diseases of Children, p. 607, American edition. Philadelphia, 1881.

in parts, in Germany, and the opinions of its numerous authors, all men of high standing, will be received with respect.*

Dr. Otto Soltmann, of Breslau, in treating of epilepsy, says (p. 103):

"The eclamptic attack cannot be distinguished by its symptoms from the epileptic attack."

Nothnagel, an authority upon the subject of epilepsy, writes as follows of eclampsia, in Ziemssen's Cyclopædia:†

"What is there now remaining of what was formerly recognized as eclampsia? Are we altogether justified in still retaining the name? We believe so, and are of opinion that the title of eclampsia should be reserved as the name of an *independent affection,* which, it is true, can at present be defined only by its clinical symptoms. We propose that the designation eclampsia should be made use of for those cases of epileptiform spasms which—independently of positive organic diseases—present themselves as an independent acute malady, and in which, so far as our present knowledge allows us to judge, the same processes arise, generally in the way of reflex excitement, and the same mechanism in the establishment of the paroxysms comes into play, as in the epileptic seizure itself. In this way, as we see, *the designation of eclampsia as an acute epilepsy finds greater authorization* [the italics are my own]; at the same time it is distinguished from true epilepsy by the lack of a persistent central change, which latter impresses upon epilepsy the character of a chronic condition. In the case of eclampsia, where this chronic change is absent, the manifestations, the seizures, disappear with the removal of the exciting irritation."

We may sum up these statements of high authorities by saying that eclamptic and epileptic attacks are similar in character and practically indistinguishable.

This being admitted as being true of the *symptoms,* we yet have the two *affections,* eclampsia and epilepsy, to differentiate; and it is this differentiation or differential diagnosis which is all-important for the welfare of our patient. It is not so very serious to consider eclampsia as epilepsy for a few months;

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* Handbuch der Kinderkrankheiten: Herausgegeben von Dr. C. Gerhardt. Bd. v., Abth. i. 1ste Hälfte: Krankheiten des Nerven-systems. Tübingen, 1879–80.

† Cyclopædia of the Practice of Medicine. Edited by Prof. H. von Ziemssen. American edition. Article on Eclampsia, vol. xiv., pp. 301–2.

but the converse mistake—the one illustrated by the cases I have read, the mistake which I believe is common, is in one sense fatal to the patient. The non-recognition of epilepsy allows of recurrence of paroxysms and the establishment of the epileptic habit.

Upon what grounds can a reasonably accurate diagnosis be made? I believe this can generally be done by attention to the physiological law of convulsibility, and to the relatively small importance of local irritations, internal and external, after the third year of life, as a cause of eclampsia. These two points have already been referred to at some length. A third rule which must be borne in mind is, that at almost any period of life uræmia may cause eclampsia. This is more especially true of young subjects who have just passed through scarlatina with nephritis, or who have had symptoms of renal disease from any cause; and also of adults—males between thirty-five and fifty—who are liable to contraction of the kidneys.

A fourth diagnostic rule is that, in adults particularly, syphilis may cause eclampsia (*i.e.*, acute, curable epilepsy).

Plumbism and alcoholism sometimes cause eclampsia, but probably in most cases by producing renal changes and uræmia.

To apply these principles to practice, let us suppose cases of first convulsions with loss of consciousness, occurring in subjects of various ages.

1. Convulsive attacks in young children under three years.

If we can exclude injury to the head, gross organic disease of the brain, and microcephaly from premature closure of the fontanelles, the attack is probably eclamptic. This probability is increased to almost a certainty if we can accurately determine the existence of sufficient systemic or local causes for the attack.

Upon this question of sufficiency of the cause, much might be said. Often the physician is satisfied with merely determining the coincidence of a fit with a local irritation, or a supposed local irritation. Soltmann * is especially emphatic in his advice to judge these coexistent conditions carefully before pronouncing them to be causes and the attacks to be merely eclamptic.

The occurrence of a single fit enhances the probability of its being the first seizure of epilepsy.

The occurrence of repeated attacks in the course of an hour or two makes it probable that the convulsions are caused by fever, by gingival, gastric, or intestinal irritation, or perhaps by some peripheral cause.

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* L. c., pp. 49–54.

2. Convulsions in young persons from three to fifteen years of age.

These are quite certainly epileptic, if we can exclude renal disease. The occurrence of attacks of an eclamptic nature (*i.e.*, ephemeral and curable) in such subjects from intestinal, or gastric, or sexual irritation, is exceedingly rare, and the mistake —the terrible mistake—of assuming such to be the pathology of convulsions, is frequently made, even by experienced physicians.

I would repeat, and the foregoing cases bear me out, that convulsions from worms, from indigestion, from lithæmia, or oxaluria, in youth are exceedingly rare, and that in the treatment of such a case the patient should be given the benefit of the doubt and be put upon a rigid anti-epileptic treatment by means of bromides, while the treatment for the supposed local or diathetic cause is being carried out.

3. Convulsions in adolescents and adults.

These are to be judged by the same general rules as No. 2, with the addition that two morbid conditions should be carefully searched for, especially when the first convulsion occurs after twenty.

a. Syphilis. This may be acquired at almost any age, but especially after sixteen or eighteen years. Nothing in the social standing of the patient should deter the physician from inquiring delicately, yet deeply, into this question.

b. Chronic interstitial nephritis, more particularly in subjects of forty years and upward. The presence of a hard pulse, of over-action and hypertrophy of the heart, the passage of an excessive amount of urine of low specific gravity, sometimes containing albumen (never much), and a few hyaline or granular casts—these symptoms go to justify the diagnosis of contracted kidneys, consequent chronic uræmia, and the occurrence of eclamptic attacks.

If we exclude these two pathological conditions, a convulsion in an adult, especially if a single fit, is quite certainly epileptic, and will be followed by others, after a lapse of time which may vary from a few days to more than a year. Of course the existence of a long interval of health after one epileptic attack in no wise justifies a physician in pronouncing the disease not to be epilepsy, as is shown by some of the cases I have read, and by numerous others which I might cite.

To sum up the early diagnosis of convulsions:

1. After the third year such attacks are very probably epileptic. The possibility of uræmia and of syphilis should be borne in mind, and a careful investigation be made as to their existence.
2. Under the third year the attack may be eclamptic—probably is—but its causes should be carefully judged.
3. In many cases under three years it is well to give a moderate amount of bromide of potassium (or sodium) with regularity for several months after a convulsion, that is to say, in such cases as do not present an evident, indisputable pathological condition sufficient to cause eclampsia.
4. In all cases above three years the bromide treatment should be at once instituted and kept up for many months.

This will not interfere with the treatment by appropriate remedies and by hygiene of gastric or intestinal indigestion, of worms, of sexual irritation, of uræmia, and of syphilis.

Besides bromides, a variety of treatment is demanded by different forms of epilepsy, according to the pathological condition; but the consideration of these indications is foreign to this paper, whose main object is to encourage the prompt and proper treatment of epilepsy at the earliest possible moment, viz., in most cases after the first attack.

I am confident that, if this were done, the prognosis of convulsive epilepsy would be greatly changed for the better.

I now pass to the consideration of the diagnosis of petit-mal, consisting of epileptic vertigo (so called), and of imperfect or aborted spasmodic seizures.

In this category I do not include the localized or hemiplegic epileptic spasms, which I have treated of in a former paper.

Petit-mal, or epileptic vertigo, is often allowed to pass for vertigo caused by indigestion. In my experience, physicians are very loath to call these slight attacks by the terrible name of epilepsy, and so delude themselves and their patients until the recurrence of a convulsive attack settles the question.

Besides, I find that, even when the attacks are recognized as epileptic, a most unfortunate statement is made that these are slight and manageable attacks, whereas the truth is that petit-mal is much more intractable than grand-mal, and often leads to more evident mental deterioration.

The correct diagnosis of petit-mal is feasible, provided a good description of the seizures be had.

From vertigo it is distinguished by:

1. The subjective phenomena. In vertigo there occurs a sensation as if the patient himself or objects about him were whirling around; in petit-mal there is no such feeling, but a sensation of confusion, or of something rising from the throat or epigastrium to the head. In some cases there are no sensations in the head beyond the consciousness that something is wrong for a moment.

The sensations of petit-mal are, moreover, usually sudden, or even flash-like, whereas in vertigo, cardiac syncope, and some hysterical attacks, there elapses quite a time in which the attack is growing. This suddenness of onset is very characteristic of minor epilepsy.

2. By objective phenomena. In faints and in some hysterical states the patient is limp from the start, and in other hysterical attacks there is spasm lasting many minutes. In petit-mal there is nearly always spasm, but not as in grand-mal. It usually expresses itself by a momentary rigidity of the whole body, with staring eyes and wide pupils. To express it otherwise, there is for an instant an unnatural immobility—the patient is, as it were, petrified for a few seconds. The friends of patients will usually accept the suggestion that the patient is statue-like in the attack.

It is to be borne in mind that in some cases the patient keeps his equilibrium, or even continues to walk. Nearly always, however, the action which the patient was doing at the moment (eating, talking, walking) is impeded or interrupted, to be resumed naturally after a few moments.

Some of these attacks of petit-mal are literally like a flash—just a moment's obscuration of consciousness. The consciousness is wholly lost in the various forms of petit-mal, though many patients will claim the contrary. The truth is usually easy to learn from the patient, or friends of the patient, and is at once evident if you happen to witness a paroxysm. I am in the habit of not relying upon an epileptic's statement that he is conscious during an attack, without sufficient corroborative testimony.

The dilatation of the pupils and their immobility, and the open state of the eyes, are capital symptoms.

In syncope and hysteria the eyes are closed and the muscles limber. The lids in hysterical "faints" present an almost pathognomonic appearance; they are rather tightly closed, and present vibrations or quivering motions due to the prolonged effort at closure. In neither of these conditions is the pupil fixed and widely dilated, as in epilepsy; this is a symptom which cannot be imitated.

Vertigo from gastric disorder is characterized by a sense of whirling in the head, and often a sensation as if the ground were opening in front of the patient, or falling away from before him, with impending precipitation. The observer notices no dilatation of the pupils, or staring, or momentary stiffness of body; the patient can speak at any time. In severe cases the vertigo is very frequent and is produced by the least motion.

I cannot enter fully into a description, for diagnostic purposes, of each and every variety of petit-mal. This would take a long time.

Allow me to refer to the intermediate attacks, in which there is some jerking of one of the limbs, or in which the patient says or does something odd. In some cases the patient will rise suddenly from a chair, walk rapidly about, muttering something. In other cases the patient will lie back in his chair with the epileptic facies, and jerk both arms or the limbs on one side of the body for a few moments. In other cases, the patient being out of doors walking in the street, loses himself for a few blocks, and is surprised at his change of location. In other cases there may be incoherent or semi-coherent talking. Other patients simply stare and make swallowing movements, with or without dreuling. Other patients fumble and fuss about with their hands, while staring and unconscious.

The unconsciousness and the attendant pupillary phenomena are the chief diagnostic symptoms in these cases; but a very important element in the differential diagnosis between these attacks and hysterical ones is that the latter present variations each time, whereas the mixed epileptic seizure is almost a stereotyped performance, one or two sets of movements being done by the unconscious subject.

Still other cases of non-spasmodic epilepsy occur in the shape of periodic or paroxysmal attacks of mania or melancholia. In some of these cases the careful observer finds that a nocturnal fit or an unobserved diurnal paroxysm ushers in the psychosis; but in other cases the mental disorder appears in a periodic epileptoid manner, and convulsions or petit-mal make their appearance later on.

I have already given it as my opinion, or rather as the summary of my experience, that petit-mal is often ignored for years, and is usually looked upon as a trivial affection.

It is my present purpose to urge the early recognition and careful treatment of this seemingly insignificant symptom. It appears to parents, and too often to physicians, as infinitely less serious than grand-mal or "fits;" yet I can assure you that the contrary is true.

Petit-mal, especially the flash-like form, is exceedingly rebellious to treatment. I have now several little patients who continue to have several "turns" a day, despite the use of as much bromide, etc., as their systems will bear. I have repeatedly had to produce severe bromism in order to barely control these minor forms of epilepsy, and any reduction of the medicine to a safer dose was followed by a return of symptoms. In taking charge of a patient who has such petit-mal I always explain to the parents or relatives the difficulty of the task they have brought to me. In my experience, spasmodic attacks—even the most severe fits—can nearly always be controlled by a proper dosing of the bromides—they may also be suspended for months and years; but we have little control of the minor manifestations of the disease.

Still, in all forms of epilepsy the date of its recognition as epilepsy is an all-important factor in prognosis. By repeated seizures a condition of the nervous system (epileptic centre?) becomes established, which we designate as the epileptic habit, a condition which explains the remarkable fact that in some cases of symptomatic or reflex epilepsy the attacks continue after removal by surgical means of the morbid focus whence the attacks seemed to be produced.

By instituting treatment very early, if possible after the first or second attack, we eliminate this factor, and the chances of cure are greatly increased.

ON THE METHODS OF DIAGNOSIS IN DISEASES OF THE NERVOUS SYSTEM.*

LECTURE I.

GENTLEMEN :—I propose to devote this and a subsequent lecture to a brief summary of the methods of diagnosis, more particularly those resorted to in organic nervous diseases, describing the instruments used and showing you how to go over a patient's body and examine it. A knowledge of the methods of diagnosis and of how much trustworthy information they will yield us is very important, and, I think, may be considered with profit. I will have a patient brought in, that I may make such manipulations upon him as are used in forming a diagnosis. Of the functions of the nervous system there are two general divisions: in the first place, the functions of movement; and in the second place, the functions of sensibility. An examination into the psychic functions is of greater importance, but hardly a subject for a clinical discourse.

For determining the condition of the functions of movement there are three methods, viz.:

1st. By means of dynamometers.

2d. By the performance of passive and active movements.

3d. By inspection.

1. The instrument which is mostly used in making examinations into disorders of movement is the dynamometer, or strength-measuring instrument (see Fig. 1). There are several varieties of dynamometers. The one I use is the simplest kind, and

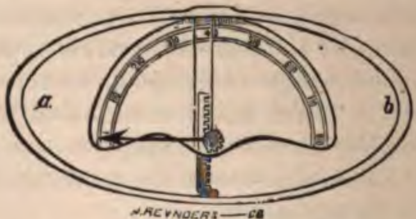

FIG. 1.

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* From the New York *Medical Record*, Dec. 3d and 24th, 1881, and *The Medical Press and Circular*, Oct. 4th, 11th and 18th, 1882.

consists of an elliptical steel spring which can be compressed in the hand or other parts of the body. Connected with it is a cog-wheel, which moves an index needle upon a dial, the index needle remaining where it is carried by the muscular effort of the patient. The French instrument is provided with a double scale, which I think is altogether unnecessary. Observations made with this dynamometer, when recorded, should be accompanied with a statement as to whether the numbers refer to the inner scale or outer scale, as the numbers indicated on the two scales differ enormously. For instance, the dynamometer needle in this patient's grasp points to 200 on the outer scale, and 70 on the inner scale. These numbers are supposed to represent kilogrammes, but I have no faith in the accuracy of such representations. Again, there is no advantage to be gained by knowing the exact number of kilogrammes of force exerted. A registration of degrees of force is entirely sufficient, and I always speak of the numbers indicated in any particular case as so many degrees. Now, as to the method of grasping the instrument. It should be grasped fairly and squarely in the hand, with the second phalanges resting upon the spring, and should be compressed without resting the hand or arm upon anything, and without giving the arm a swing at the moment of compression, as some patients will do. It is also important to place the face of the dial toward the palm of the hand, otherwise the fingers of the patient may interfere with the movement of the index needle. We know that there is in most healthy individuals a difference in strength between the left and right hands, but we want to estimate it. There are very few healthy individuals with a difference of more than five or eight degrees; a difference of ten degrees would lead to the suspicion of paralytic weakness upon one side. Sometimes we find the left hand stronger than the right. A number of patients are left-handed, and therefore use the left hand more than the right. There are some occupations which tend to develop more strength in the left hand than in the right. Car drivers show equal strength of both hands, or greater strength with the left hand, for the reason that the reins are held in it. I need not go into further details, but will say that you should be on the look-out for these normal variations in making tests with the dynamometer.

My friend, Dr. Birdsall, has invented a foot dynamometer, which is intended to record the comparative degrees of strength of either the anterior or posterior tibial group of muscles upon the two sides of the body. It consists (see Fig. 2) of a board with a movable foot-piece, which is grooved so as to receive the hand dynamometer between the base-board and the movable foot-board. The foot-board is provided with a toe and heel strap, by means of which the foot is secured in position upon it. By placing the hand dynamometer in the front grooves and strapping the heel down upon the foot-board, and telling the patient to lift his heel, the strength of the posterior leg-muscles can be approximately estimated. For testing the anterior tibial group of muscles the ends of the instrument are reversed, the hand dynamometer being left in position, and the toes strapped to the foot-board. The patient is then directed to attempt to raise his toes, and in so doing the power expended is transmitted through the heel to the hand dynamometer. This instrument was brought to the notice of the profession by Dr. Birdsall, in a paper read before the American Neurological Association. Its accuracy is not as great as that of the hand dynamometer, but, as I have already remarked, we do not care so much for an accurate record of the force expended as we do for a comparative measurement of the force exerted on the two sides of the body.

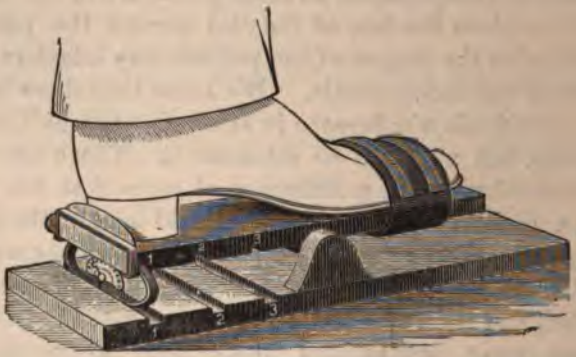

FIG. 2.

There are registering dynamometers which record upon paper, or other prepared material, the muscular strength and tone of the patient. There are also dynamometers which will register the amount of pulling or pushing force exerted, and so on; but, for practical work, all that is necessary is a good hand dynamometer.

2. Another method of detecting disorders of movement is by handling the patient. The manipulations are made with our hands, and against the patient's will. We are thus enabled to determine the amount of resistance—strength of a great many muscular groups in the body. We lay hold of the patient's limb, having directed him to hold it in a given position, and tell him to resist our efforts at flexion or extension. By having the patient resist downward pressure upon the chin, we can determine whether there be paralysis of the masseters, temporals, or pterygoids. If paralysis exist, a very slight downward pressure will be sufficient to overcome the muscular resistance, and thus open the mouth. We can judge of the condition of the neck-muscles by the movements of the head. We direct the patient to hold his head perfectly stiff and upright, and see how much force is required to move it forward or backward. In a similar way we test the power of the flexors and the extensors of the arm. In partial lead-paralysis only a very slight force is required to flex the hand. The short flexors of the hand can be tested in this way, and even the interossei can be tested by lateral movements of the fingers. With the lower extremities we proceed in a corresponding way. To test the quadriceps extensor femoris, we make the patient hold the leg in extension and attempt to flex it. For testing the posterior thigh muscles we cause the patient to flex the leg upon the thigh, and take hold of the foot and pull it forward. The same test which was applied to the hands and fingers can be applied to the foot and toes. The abductors and adductors should be tested in the same way. In little children under three years the condition of the muscles may be examined by what I am in the habit of calling the cataleptoid test; or, more properly speaking, we note the absence or presence of the cataleptoid state in the muscles. I have found that if we divert the attention of a healthy baby and gently place one of its arms or legs in a given position, or in any position, that position will be maintained for several seconds or minutes. The limb is held by an automatic muscular contraction—an exaggeration of the muscular tonus. This normal state I call the cataleptoid state. In paresis or paralysis, more especially in infantile spinal paralysis, the affected limb, when most

carefully placed as described above, falls at once, like lead, in obedience with gravity.

The ocular muscles can be tested by requesting the patient to follow an object about with his eyes without moving his head. Convergent and lateral movements can thus be studied. A good way of testing the interni is to hold your finger at about twelve inches from and in front of the patient's eyes, and move it gradually to within three inches. If affected, one eye or both eyes will diverge. We test the external recti muscles by making the patient follow an object in the outer fields of vision. It must not be assumed in obscure cases that this test is sufficient. There are cases of diplopia which are not revealed by this means. The method by which the oculists determine the existence of latent diplopia is to hold a strip of colored glass before one eye so as to color one of the images of the object, and thus enable the patient to distinguish them. Prisms are also used for detecting slight weakness of the recti muscles; but, as this is a part of a delicate manipulation of special eye practice, I need not more than refer to it.

3. The third method is by observing the patient's movements, or by inspection. The tongue is tested by requesting the patient to protrude it, and observing whether it deviates to the right or left, and how far it is protruded. You should guard against error in making the projection test. If the patient has lost a good many teeth upon one side, the tongue may be deviated in consequence, or it may be turned out of line by a cicatrix in itself. The tongue may be deviated by paralysis of one cheek. These sources of error are of considerable importance in a practical way. Facial paralysis on one side is evidenced by a drawing of the features to the opposite side, as well as by loss of expression on the affected side. In paralysis of the orbicularis palpebrarum the eye cannot be closed, etc. The various movements of the arm are to be tried in a systematic way. In evident paralysis, of course, we do not make these tests, but in slight cases we tell the patient to move both arms simultaneously, and note which one moves slower and less forcibly. I will show you a case of ordinary hemiplegia. I ask the patient to hold up both arms, but you see that the paralyzed arm does not go up like the other.

By voluntary movements we test the various forms of incoordination. We test co-ordination by testing the precision of the movements of the muscles, not their strength. A ready means of doing this for the arm is to request the patient to shut his eyes and try to put the tip of his forefinger upon the end of his nose. Some patients cannot do this. In that form of sclerosis known as disseminated cerebro-spinal sclerosis, and in posterior spinal sclerosis involving the cervical cord, the patient, upon being submitted to the above test, has oscillating movements of the arm and finger, and ends by thrusting his finger into his eyes or mouth. Similar oscillations are shown when the patient attempts to put a key in a key-hole. These resemble closely the movements of a person trying to do the same thing while in a state of acute alcoholic intoxication.

The movements of the legs and feet in walking have been the subject of much study. Some observers have endeavored to record the peculiarities of gait by means of giant sheets of papers so prepared that when a patient walks upon them imprints of his footsteps will be left. Such tests require considerable expenditure of time, and hence I would ask you to learn to judge of the gait of a patient by inspection and by the sense of hearing.

In the hemiplegic gait the foot is turned in, the heel raised, and the patient brings the extremity forward with a swinging, semicircular movement; he cannot bend the knee properly. This is what the French call the sickle walk. As the foot is brought forward, the outer portion of the sole of the shoe scrapes upon the floor. Hence it is that these patients first wear out the soles of their shoes upon the outer side. As a part of the hemiplegic gait should be noted the drooping shoulder of the affected side.

A characteristic walk is the ataxic. In this the leg is well raised, and the patient overdoes the extension and abduction movements of the step, bringing the foot down, heel first, in a jerking manner. A good method of testing co-ordination of the legs is by driving a pin into the toe of the patient's boot, and requesting him, while seated, to approximate the head of that pin to the head of another which you hold above it. If there be ataxia, the foot will oscillate in this movement, as the finger does in the nose test. In the recumbent posture there is a more classical test of ataxia. Covering the patient's eyes, we bid him put his right heel upon his left patella. If he be ataxic, the heel wanders about in a vain attempt to hit the mark.

Another peculiar walk, which is of considerable frequency, is the paraplegic, the walk of partial paralysis. In chronic transverse myelitis the patient walks with a scuffling movement, dragging his toes upon the floor.

By closing your eyes and listening to the walk of two patients, one ataxic and the other paraplegic, you can tell them apart easily. The former makes a loud stamping sound with his heels; the latter scrapes the floor with his feet.

An important walk is the tetanoid, which I first described in 1873.[*] The patient takes very small steps and turns the toes inward. The feet are approximated, and there is a tendency to crossing of the legs from overaction of the adductors. The legs are rigid and the feet drag. You will find this in many children, occasionally in adults. This walk is believed to indicate primary or secondary sclerosis of the lateral columns, or want of development of the same parts.

In hysterical hemiplegia the walk is peculiar. A woman may seem to have complete paralysis of the arm and partial paralysis of one leg. In walking she moves the leg in the proper vertical line; there is no sickle movement; the foot is carried carefully and squarely over the ground, and is dragged. Dr. R. B. Todd,[†] of London, who was one of the most acute and skilful students of nervous diseases thirty years ago, first described this walk.

The walk of patients with cerebellar disease is usually badly denominated in the books. It is spoken of as ataxic, but there is nothing ataxic about it. If you were to test a patient with cerebellar disease as you would one with ataxia, by requesting him to shut his eyes and put the end of his forefinger upon the end of his nose, etc., he would have no difficulty in doing it. The walk of cerebellar disease is inco-ordinate, though not ataxic. It is more like the inco-ordination of intoxication, or, as the French writers call it, *titubating*. The patient tries to extend his base of support by separating his feet, and his body oscillates from side to side. If the feet be bare, the patient's toes will be seen working abnormally, and apparently digging into the carpet. Nothnagel[‡] has written the best account of the cerebellar walk.

I will add a few words about reflexes. These are tested usually by pinching, burning, or tickling the soles of the feet and other parts of the body. The tendinous reflexes are of more recent discovery. We determine their existence or non-existence by tapping upon the tendons with the finger, as in percussion. Then, there are internal or visceral reflexes, such as those of the bladder resulting from the organ being filled with urine, those of the pharynx, and so on. In most of our examinations we test the tendinous reflexes. The tendon reflex can be tested anywhere that a tendon can be got at. Upon the leg you can tap the tendo Achillis. The most commonly used and most convenient place of testing the tendon reflex is at the knee. If we tap upon the hamstring tendons we get a movement backward. The tendon of the quadriceps extensor femoris, or ligamentum patellæ is always an object of investigation, and striking it causes a partial extension of the leg. In certain spinal diseases no reflex movement is there present; in other conditions of the cord there is increased reflex at the knee. The best way of judging this is to use at first a minimum amount of force in tapping the tendon, increasing it as necessary to develop the reflex movement. Dr. Gray, of Brooklyn, made some interesting experiments three years ago upon some of the students of the Long Island Hospital Medical College as to the normal tendon-reflex at the knee. He found it absent in two or three individuals out of a hundred healthy subjects; and I have seen similar exceptions.

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[*] "Description of a Peculiar Paraplegiform Affection." See p. 127. Also *Archives of Scientific and Practical Medicine*, No. 2. New York, February, 1873, vol. i., p. 101.

[†] "Clinical Lectures on Paralysis," etc. London, 1856, p. 257.

[‡] "Topische Diagnostik der Gehirn-Krankheiten." Berlin, 1879, pp. 59–51.

Disorders of sensibility.—Here we come in contact with the consciousness of the patient—the ego of the patient—and we are to a certain extent at his mercy. In children this part of the diagnosis is limited to a very great degree on account of their inferior intelligence. For example, the sense of sight cannot be fully tested in these little patients, and when testing common sensibility we have to pinch and tickle them, and make use of other coarse tests. The special examination of the eyes and ears I will pass over for the present.

We will now consider the perception and localization of simple contact such as produced by the finger or a feather. The patient's eyes being closed, we touch him slightly in different parts, and ask him if he feels, and where he feels the contact.

The question of "muscular sense" is an involved one. Some believe that the muscles have no proper sensibility, and that we know our muscular movements by psychic estimation of the force employed, yet there are some persons who cannot feel a tap or prick upon the skin of the leg, yet will feel firm pressure made upon the calf. There have been instruments devised for determining the amount of weight in grammes which a patient can distinguish. We test sensibility to pain in various ways. The use of the hot iron and matches I object to. We are not allowed, in an examination, to adopt such methods as will result in scars. A faradic wire brush is harmless, produces no scar, and is really more painful than cutting or burning, as I have repeatedly seen; and a clean, sharp needle is not objectionable.

The brothers Weber, of Leipsig, gave us forty years ago a form of æsthesiometer, which is much like a shoemaker's rule, with one end stationary.

Another form of æsthesiometer is made like a compass, with or without a vernier. All æsthesiometers should have dull points. Dr. Birdsall has devised an improvement upon the former instrument. It consists in the attachment of a point at one end of the instrument, so that a patient can be pricked at will. We know from Weber's researches the distance at which two points of the æsthesiometer can be distinguished on all the parts of the body. The following are some of the more important according to Valentin:*

	Millimetres.
At the tip of the tongue	1.00
" palmar surface of tips of fingers	1.50
" " " second phalanges	3.24
" " " first phalanges	3.44
" dorsum of the tongue	5.22
" dorsal surface of fingers	8.12
" cheek	9.46
" back of hand	14.50
" skin of throat	17.27
" dorsum of foot	26.10
" front of sternum	33.07
" middle of back	50.43

In estimating pathological conditions I am in the habit of making considerable allowance for normal variations. The points are to be applied to the surface of the skin simultaneously. This requires a certain degree of expertness on the part of an operator, and in order that the translation of the impressions be correct, some degree of intelligence on the part of the patient is required. In testing the tongue, the patient is to indicate the answer by holding up one or two fingers. With the thumb and forefinger you can increase or decrease the distance between the points.

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* For a complete table in lines (about 2 mm.) consult Flint's Text-Book of Physiology, p. 753. New York, 1881.

One word about motor and sensory tests with a battery. For this purpose, either a faradic or galvanic current can be used. We use both currents for testing the condition of the connection between the nervous centres and the peripheral organs. I can only formulate a general law, and it is this: That in cases of disease of the brain in which the spinal cord and nerves continue to act upon the muscles, even though the will has no influence over them, the electrical reactions are preserved. In cases of certain spinal lesions, especially in transverse lesions, the same law holds true.

The affections of the spinal cord which destroy or involve the gray matter of the anterior horns will be accompanied, within a week or two weeks, by a loss of reaction in the paralyzed muscles and their nerves; whereas, with a lesion of the spinal cord which leaves a certain part of the gray matter below the lesion uninjured, even though it be but a centimetre, we will get reactions in those muscles connected with the healthy part of the cord. In cases of injury to the spinal cord very low down, which destroy the lumbar enlargement, the muscles of the paralyzed legs undergo atrophy and lose their reaction to faradism. With a destructive lesion of the cervical enlargement, the same occurs in the hand and arm. The law may be stated in the following words: Destructive lesions of the gray matter of the cord abolish faradic reaction in the associated or dependent muscles. The same law is applicable to so-called peripheral paralysis, in which the nerve trunk has been divided or crushed. Hence, you see there is nothing pathognomonic determined by means of the electrical test; it is of conditional value.

I would like to say something about the use of the ophthalmoscope. I consider its use of great importance in the study of nervous diseases; yet I feel that too much has recently been claimed for it. I would urge all of you to learn its use and to recognize the healthy fundus. By so doing you will get negative information; and you should also learn to recognize atrophy of the optic nerve, neuro-retinitis, or choked disc, and the retinitis of Bright's disease. These signs are positive and valuable indications of disease. It has been claimed that by the ophthalmoscope we can determine the condition of the intra-cranial circulation, judging of this by the state of the retinal vessels. This I do not believe to be possible, partly because of the great normal variation in the circulation at the bottom of the eye; partly because of the varying appearances of the vessels, according to the condition of the refraction of the eye; and lastly, because it is not at all certain that there is any correspondence between the retinal and cerebral circulations. I am supported in these statements by leading oculists, who consider it a delicate task to decide whether a retina be hyperæmic or anæmic.

LECTURE II.

GENTLEMEN:—In my last lecture I spoke of the objective part of a diagnosis, and pointed out in brief the principal disturbances of function which we are able to recognize in an examination, and exhibited and described the instruments by means of which we detected and measured the degree of such disturbances. I referred to certain methods of diagnosis which fall within the province of other specialties, more particularly the use of the ophthalmoscope. To-day I wish to extend my remarks to certain other methods of diagnosis, a knowledge of which is essential to the success of the nervous specialist. Among these may be mentioned auscultation and percussion in the physical examination of the chest and abdomen, the use of specula and tuning-forks in the examination of the ear and testing the hearing, the use of the laryngoscope in the examination of the larynx, the chemical and microscopical examination of the urine, and so on.

Of course the neurologist cannot be expert in all these methods of examination; but, on the other hand, he cannot be expected to make a satisfactory study of his patients without a certain degree of skill in these diagnostic manœuvres. With respect to urine, it is especially important that he should be able to make a satisfactory examination of it. I may say that I have often had occasion, during the past five or six years, to prove the dependence of an obstinate and severe form of headache upon the existence of contracted kidneys. Detecting the presence of an excess of phosphates, or of an excess of urea, or the condition known as oxaluria, is often a very important factor in nervous diagnosis. It is not sufficient to make a single examination of a sample of urine in a test-tube, by boiling it and adding acid, but the amount of urine secreted in twenty-four hours should be known, and, from repeated examinations of it, the existence or non-existence of morbid conditions noted.

By an examination of the genital organs, both male and female, much important information may be derived.

A knowledge of the presence of versions, flexions, or tumors of the womb, ovarian disease, deep-seated stricture, or narrow anterior urethra, and many other abnormal conditions of the genital tract, will lead to the adoption of appropriate treatment; whereas, without such knowledge, drugs may be prescribed and electric batteries used, for an indefinite time, without relief.

I next propose to speak of the principles of diagnosis, and will divide the disorders to be diagnosticated into two classes, organic and functional. This division presupposes a difference in the method of examination. The logical difference between the methods of study is very marked. The complete and satisfactory recognition of organic disease of the nervous system involves a triple diagnosis. It is arrived at inductively by means of the history given by the patient, by means of the testimony of friends, by means of the various methods of physical examination, and, to a certain extent, deductively by knowledge you have acquired from experience, books, etc.

The first diagnosis is that of the symptoms, and these may be considered as constituting what the Germans call the "symptom group." Second, we make a diagnosis of the location of the lesion, and it is this diagnosis which is just now interesting observers most. In the third place, we have to determine the nature of the lesion, which is, perhaps, of the greatest practical importance to the physician. We first determine the symptoms, next the situation of the lesion producing the symptoms, and afterward try to determine the pathological nature of the lesion.

The first diagnosis is made exclusively from a history of the case and an objective examination. We learn from the statements of the patient and friends, and an ocular examination, what has taken place during the few days or weeks preceding. We determine, by methods pointed out in the last lecture, whether there exists paralysis, spasm, ataxia, anæsthesia, etc. The mental symptoms to be noted are loss of memory, delusions, or uncontrollable manifestations. We thoroughly overhaul the patient physically and psychically, and we determine what functions are well performed, and which are interfered with. During the next few weeks you will have in every lecture an illustration of how to make this diagnosis: for example, a patient will come into the clinic complaining of weakness of the muscles of the face, arm, and leg, upon one side of the body.

We determine the existence of this condition by the testimony of the patient and his friends, by inspecting the patient, by the application of tests which have been already described to you, and from the data thus collected decide that the case is properly one of hemiplegia. Again, a patient may come in complaining of weakness of both legs, while the arms are normal, and we decide that he is suffering from paraplegia. A patient presenting with no paralysis and no anæsthesia, but with an inability to direct the movements of his legs, we say he is suffering from inco-ordination or ataxia. If a patient presents with one eye turned outward and a drooping of the lid, we designate this condition as one of ptosis with strabismus. If a patient has loss of sensibility, we call it anæsthesia, and ascribe particular names, in accordance with the distribution of the anæsthesia. When anæsthesia of one-half of the body exists, we call it hemi-anæsthesia. If, upon examination, vision is found to exist only in one-half of the visual field, we call the condition one of hemiopia. In this manner we indicate the condition of the various organs throughout the body, and determine in what way they differ from the normal, and to the conditions found to exist we give certain names which represent the symptom-group.

Sometimes we are only able to discover a single symptom, and have to go on to the next diagnosis. My observations have led me to believe that in making the first diagnosis, the testimony of the patient is too frequently used as a sufficient guide, and pains are not taken to go over the body and carefully determine the extent of paralysis, ataxia, or areas of anæsthesia, and make a record of what is found. There is often a lack a definiteness in examinations of nervous cases which practitioners would not allow in chest diseases. From the time of Läenec no physician has considered his examination of the chest complete without having determined the nature and character of the signs, so that he could tell another physician what he had seen or heard, in such a way as to lead him to a correct diagnosis. We are attempting to apply the same exact method to the study of nervous diseases, and are making very great progress.

The second diagnosis, that of the location of the lesion, is of importance. It is not directly dependent upon observations made with our eyes, instruments, etc., but upon anatomical and physiological knowledge. It is very seldom that a neural lesion is visible, except in cases resulting from injury or external violence. We have to determine the situation of the lesion from the symptoms present, by means of our acquired knowledge of physiology and anatomy. Hence it is that exact and minute anatomical knowledge is more essential for the diagnosis of nervous diseases than for that of any other.

I will take up some of the symptom groups of which I spoke a few moments ago, as determined hypothetically in my remarks: for example, that of hemiplegia. We revert to our acquired knowledge concerning the nervous supply of the paralyzed parts derived from the spinal cord and brain. We also revert to the experiments made upon the central nervous system of higher animals within the last fifteen years, and our empirical knowledge of lesions discovered after death in cases of hemiplegia studied during life. For the present I will rely more particularly upon anatomical and experimental data. We know that the limbs of one side of the body receive innervation from the opposite side of the brain. You will hear it said that this law is not valid; but I say to you it is a good law, and a knowledge of it will be of great help to you in the study of nervous diseases. The few exceptions which are quoted in support of the idea of its invalidity are capable of many interpretations which leave the law untouched for all practical purposes. I propose to discuss this matter of the crossing of nerve fibres from one side to the other, in a subsequent lecture. Bearing in mind, then, that either side of the body is innervated from the opposite side of the brain, we at once locate the lesion on the opposite side from that in which the paralysis is found to be present in our hypothetical case of hemiplegia. Partly from the study of anatomy, partly from the study of embryology, and partly from a knowledge of what recent experimental physiology has taught, we know that the whole of one side of the brain has not the power of moving all the opposite side of the body. Let us suppose a case of left hemiplegia with the lesion in the right hemisphere. I may remark that the whole right hemisphere is not connected with the opposite cheek, arm, and foot. Recent experiments have shown that a relatively small part of the right hemisphere has power over the left cheek, hand, and leg, and this part of the hemisphere is in the median region, and embraces a small number of convolutions—certainly not more than half of them. We have records of positive knowledge, to the effect that the anterior and posterior extremities of the hemispheres have no motor power over the opposite side. Still basing ourselves upon anatomical researches and physiological experiments, we know that the muscles of the left side of the face are innervated by a small part of the right frontal lobe, we know that the upper extremity is innervated by a small part of the brain known as the ascending frontal and parietal convolutions. We further note that the part of this right hemisphere which is connected with the leg is situated at a different point, nearer the median line, near and in the longitudinal fissure, and more posteriorly. In other words, we have determined with great accuracy the motor area of the right hemisphere, and the centres for the movements of the cheek, arm, and leg. With respect to the sensory function of the hemisphere our knowledge is not so exact, but we know that loss of sensibility on one side of the body indicates a lesion in the opposite half of the brain; that right hemi-anæsthesia indicates a lesion in the left hemisphere—one situated in all probability in parts posterior to the motor area. These are not theoretical guides; they are valuable life-laws, and there has not a year passed, within the last five years, that I have not seen cases illustrative of them, and verified them by post-mortem examination. Bearing upon this point, I may say that I published two cases this year,* in which the diagnosis was made, by means of a knowledge of these laws, with such accuracy that we were within 1.5 to 3. centimetres of the actual seat of the lesion. In a number of my cases the lesion was found to be situated exactly where it was diagnosticated during life. Records of similar cases may be found in American, English, and Continental journals generally.

We are also able to locate, upon similar anatomical and physiological principles, lesions of the spinal cord. In paraplegia we can determine the height of the lesion quite accurately, by means of our knowledge of the points at which nerves are given off trom the spinal cord. To quote a case: a patient falls and receives a dislocation and fracture of the seventh cervical vertebra, compressing the cord at this point. The patient at once presents anæsthesia and paralysis below the clavicles and in the distribution of the ulnar nerve. Now we know, from anatomical and some curious electrical * examinations, that the fibres of the ulnar nerve arise from the lower part of the cervical enlargement, and that its trunk is made up by the last cervical and first dorsal nerves. Hence it is that compression of the cord

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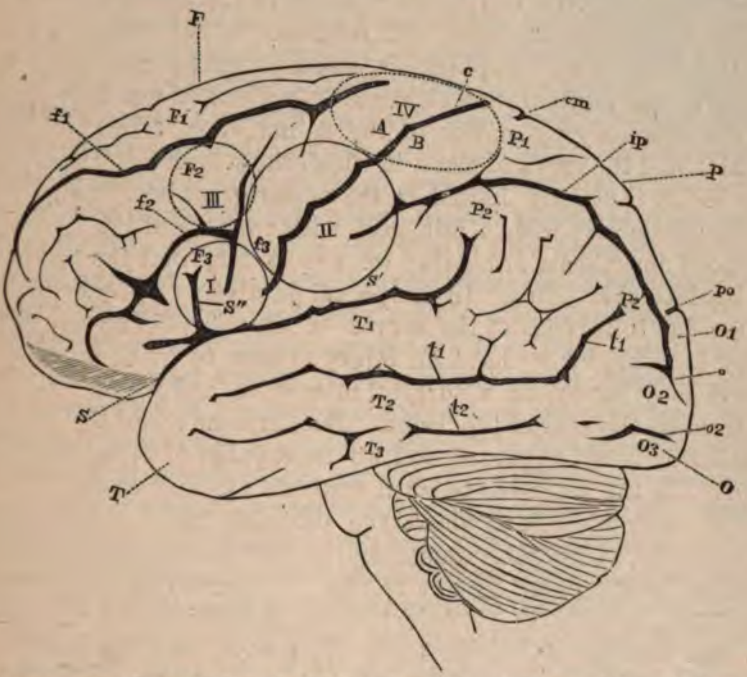
* *Journal of Nervous and Mental Diseases*, July, 1881, p. 510, vol. viii. Also p. . . . of this volume.



The above woodcut is modified from Ferrier : letters and figures the same.—S, fissure of Sylvius ; c, fissure of Rolando ; po, parieto-occipital fissure. A, ascending frontal gyrus ; B, ascending parietal gyrus ; F_3, third frontal gyrus ; P_2, gyrus angularis. Circle I., seat of lesions which (on the left side) cause aphasia. Circle II., seat of lesions which convulse or paralyze the upper extremity of the opposite side. Dotted Circle III., seat of lesions which probably convulse or paralyze the face on the opposite side. Dotted Oval IV., seat of lesions which convulse or paralyze the lower extremity of the opposite side. These districts receive their blood supply from the middle cerebral artery.

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* In two or three cases of spinal irritation I have noticed that placing the cathode over the sixth and seventh dorsal vertebræ, with the anode near by upon an indifferent part, and passing a strong current, the patient, though devoid of any anatomical knowledge, has described a pricking sensation in the little and ring fingers of each hand. This is evidence of the cathodal excitation of the origin of the ulnar nerve.

at this point gives rise to paralyses of parts supplied by this nerve. When I come to speak of the diseases of the spinal cord, I will give in detail the rules for determining the height of any lesion, by means of which a diagnosis of extreme accuracy can now be made. We can locate a lesion in the anterior and posterior portions of the cord, with reference to the white and gray matter. We know that paralysis, accompanied by great muscular atrophy and degenerative electrical reactions, is due to a lesion in the anterior gray matter of the spinal cord. We know that in ataxia of the legs and arms, in which there is no true paralysis, the lesion is strictly limited to the posterior white columns. A patient comes in with a stiff gait, with crossing of the legs, but without anæsthesia or ataxia; we are certain that the lesion is in the lateral columns. In peripheral paralysis—as, for example, when due to injuries of the nerves—a diagnosis is usually very easy to make, for we have an injury or scar to guide us. Or a nerve may be cut in a surgical manœuvre, and we can at once determine the seat of the lesion. But, in some cases, the seat of the lesion is not apparent, and we must judge of it by medical logic. In the diagnosis of such cases we are very much helped by an anatomical law, which you probably have not found in your books, and which summarizes the distribution of the sensory and motor filaments of nerve trunks. I refer to the law of Van der Kolk, namely, that a spinal nerve gives its motor branches to the muscles as instruments of motion, and its sensitive branches to the part moved.* In other words, throughout the body the sensitive branches of a mixed nerve run to the part of the skin which is moved by the muscles receiving motor filaments from the same nerve trunk. It is very easy to call to mind examples of this law. A sciatic nerve, for example, sends its sensory filaments to the calf of the leg and the foot, which parts are moved by the posterior thigh muscles and both sets of tibial muscles, which receive their motor filaments from the sciatic. In the arm the musculo-cutaneous nerve gives motor fibres to the biceps and brachialus anticus, and its sensory filaments go to the integument of the forearm, which is the part moved by the above-named muscles.

Wherever you study this law you will find it absolute. You may meet with some apparent exceptions, but they are only apparent. An apparent exception is found in the head, where the trigeminus plays the part of sensory roots for most of the cranial motor nerves. Studied in the light of this law, a patient presenting anæsthesia of the little and ring fingers, and outer part of the palm, with paralysis of the interossei and inner half of the thenar group, has a lesion of the ulnar nerve, probably above the wrist. If the flexor carpi ulnaris is paralyzed, the lesion is certainly above the elbow. Again, paralysis of the extensors of the carpus and fingers, with anæsthesia of the dorsum of the hand on its radial side, indicates a lesion of the musculo-spinal nerve.

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* Schroeder Van der Kolk: "On the Minute Structure and Functions of the Spinal Cord and Medulla Oblongata." Translation of the New Sydenham Society, pp. 6, 7. London, 1859.

We now come to the third diagnosis—that of the nature of the lesion. This should not be made until the others have been determined carefully; and, here again, we use entirely different principles of logic. This diagnosis is more empirical—more uncertain. The third diagnosis can be best made by the physician who has had the greatest experience with disease in general; and here comes in the strongest argument for the postponement of special practice until you have acquired a thorough knowledge of general pathology. It is just here that one realizes the insufficiency of normal anatomical and physiological study. It makes no difference what the symptoms are, or where the lesion is located, for that does not determine what the lesion is. Take, for example, a left hemiplegia with paralysis of the left cheek, arm, and leg. The patient may have a tumor, a patch of softening, a hemorrhagic focus, a syphilitic formation; he may have what is exceedingly rare—local anæmia, and the only way of determining which one of these is present is by our knowledge of pathology. To this end we determine the temperature, pulse, and other symptoms; the conditions of the arteries; the presence or absence of the signs of degeneration throughout the body, and the presence or absence of the signs of contracted kidneys and heart disease. To determine with certainty that a hemorrhage has taken place in the brain, you must ascertain the previous diathesis and the general condition of the body. The rupture of vessels in the brain is a mere accident; the same statement may be made in regard to softening. This latter condition is often due to organic cardiac disease, producing embolism of a healthy cerebral artery, and sometimes also of arteries at the periphery, which is more difficult to make out. It is likewise caused by local obstructions in the arteries of the aged or prematurely old, and of syphilitic subjects. The same general rules apply to the study of diseases of the spinal cord. The nature of the lesion is to be determined by the pathological condition of the patient, and by means of empirical knowledge acquired by post-mortem examinations. For example, if a child presents with enlargement of the head and separation of the sutures, we know, as a matter of experience and post-mortem examination, that there is internal hydrocephalus, the most common cause of which is tubercle; and we also say that it is probable that the tubercle is compressing the aquæductus Sylvii. In the same way we determine the difference between syphilitic and non-syphilitic subjects.

I go on, now, to the diagnosis of the functional disorders of the nervous system. This subject does not admit of as systematic a presentation as the former. In many of the functional diseases of the nervous system we know very little about their pathology, so we do not complete the third diagnosis; and as the disease is functional, we need not consider the second. For example, chorea is an obvious symptom which we can recognise at a glance. If we attempt to localize the lesion or disease, we cannot go any farther than to make the statement that a functional lesion is situated in the brain, and that it is very probably a cerebral affection. We make this statement upon the remarkable fact that the disease is often hemiplegiform. We cannot go any farther, and, as regards the lesion of chorea, we are entirely in the dark. We can only offer speculations based upon a few post-mortem examinations, which, in my opinion, are worth very little. The same is true of epilepsy, hysteria, and neuralgia. The best we can do is to state the symptoms definitely, and not attempt any rigorous diagnosis of the disorder, or try to determine the nature of the lesion, in the same sense as we do lesions in organic affections. But yet there is a diagnosis to be made of the nature of the lesion, or, as I would put it, the condition of the patient. This consists, after we have determined that the patient has chorea, epilepsy, or neuralgia, or "neurasthenia," of making an inquiry into his general condition, the state of the circulation, arterial tension, and, in some cases, the action of the heart, and the condition and mode of action of certain organs which we know, from empirical knowledge, to be connected with functional disorders. For example, within the last seven years we have learned, with great positiveness, that certain cases of headache are due to straining of the eyes. In studying cases of headache, especially cases of long standing, it is desirable—I may say imperatively necessary—to study the condition of the eyes. Thanks to the labors of Drs. Weir Mitchell and William Thomson, of Philadelphia, we have been able to relieve a large number of cases of "bad head," as I call them. In the same way, with reference to hysteria, we examine the patient, not simply for anæmia and mal-nutrition, but also for uterine and ovarian disease or vulvar irritation. This will lead us many times to a correct understanding of the case. There is quite a large class of cases in which the symptoms are nervous, the patients experiencing a curious depression, pain in the back and head, aching in the limbs, and many other symptoms, chiefly sensory, all of which we are generally unable to refer to any definite cause, and they are known under the name of "neurasthenia." This name is all very well, provided we go farther. One theory as to the cause of these symptoms—and it is held by some very respectable authorities—is that they are due to a variation in the amount of blood in the spinal cord and brain. I myself confess to very little faith in this view. I think it can be shown that there can be but slight changes in the amount of blood present in the spinal cord, and that these are not accompanied with symptoms. My own experience is that the symptoms are often produced by conditions of general mal-nutrition, which can be classified conveniently under the heads of lithæmia, or sub-oxidation of the tissues. Some other cases are due to weak eyes, some to ovarian diseases, some to the use of tobacco, sexual excesses, etc. Cases of so-called hyperæmia of the brain, now so fashionable, are to be studied in the same way from the standpoint of the general physician; nearly all such cases being, according to my experience, resolvable into instances of lithæmia, eye-strain, cardiac disease, renal disease, irritation from the sexual organs, etc., leaving exceedingly few cases of hyperæmia, if any. The diagnosis of lithæmia is to be obtained by the examination of the urine, and the usual method of doing this is very misleading. If the patient is told to bring a bottle of his urine, the first that is passed in the morning, it is found of high specific gravity, high color, acts upon litmus paper forcibly, and may deposit urates; or the microscope shows, in a few hours, oxalate of lime.

To judge in this way from one specimen, I believe to be a most superficial practice. I am not willing to say that a patient is a subject of lithæmia or oxaluria, unless I have examined specimens of urine on successive days, for the reason that the urine varies from day to day, and during each day, owing to the varying conditions of the body. One or two examinations are of very little help. In my practice I make a number of examinations—three or four—of urine passed morning, noon, evening, and at bed-time; and I am unwilling to use the term oxaluria unless I find this condition indicated by most of the specimens. By this means the average condition of the urine in twenty-four hours can be determined.

I will make a few remarks in regard to taking the history of a case. Very few medical men take good notes. It is a very difficult matter to write a history so that different medical men, reading it, would come to the same conclusion in regard to the case. Of course we first obtain the patient's story. We have to guide the patient, so that we can arrange the facts which he gives us in some logical order. It is desirable, in many cases, to give some illustration as to the behavior of the patient, mental manifestation, etc. A few words with the friends of the patient, as to what they have observed, is often serviceable, and sometimes it is well to question the patient alone. In taking down the notes we form a dim mental outline of the diagnosis, in advance. It seems impossible to take a history without doing this. We next carefully record the objective symptoms presented by the patient, and thus reach what I have termed the first diagnosis. In some cases we may locate the lesion at once from the symptoms, and even determine the nature of the lesion. This is by no means infrequent, and the taking of the history will be the only time you need to give to the consideration of the case. In other instances you have to go on, step by step, and carefully determine the points in the diagnosis which I have referred to. We are often, in the course of this preliminary and semi-conscious notion of the nature of the case, led to doubt the patient's statements, and it is well to make a mental memorandum of these points, and, after the history is completed, question the patient again, or ask information of others. This is very necessary in cases where we investigate such causes as sexual irregularities and syphilis, family taints, etc.

THE EFFICACY OF IODIDE OF POTASSIUM IN NON-SYPHILITIC ORGANIC DISEASES OF THE CENTRAL NERVOUS SYSTEM.*

I INTEND this paper to be inquiring and suggestive rather than didatic, and hope that it may be the means of eliciting the experience and opinion of others whose opportunity for observation has been greater than my own.

There has appeared to be in the minds of those members of our profession whom I have had the pleasure of knowing, a half avowed belief in the *specific* action of potassium iodide; that it is a sort of reagent with respect to syphilis. Many go so far as to assume this position: that if an individual present a given symptom, but denies having had syphilis in any form, and if that symptom disappear under the use of the iodide of potassium, then the symptom must have been syphilitic in spite of the patient's denial. I have repeatedly heard medical teachers say of a symptom: "Give iodide, and we will see if it is syphilitic or not," the implication being that if non-syphilitic the symptom would not be removed by the drug.

The consequences of such a belief may be serious. On the one hand, a physician holding the above views will be indisposed to try the drug in full doses in cases of organic cerebral disease where there is no indication of syphilis; a negative position which might cost the lives of several patients in the course of the physician's life.

On the other hand, after curing certain symptoms with iodide of potassium in a person who claims never to have had syphilis, the physician becomes convinced that the patient has consciously or unconsciously deceived him; that he is syphilitic. Such a view will powerfully influence the physician in his further relations with the same patient as regards his interpretation and treatment of other affections which may show themselves, and in respect to the advice to be given as to marriage, child-bearing, etc.

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* Read before the New York Neurological Society, January 3, 1882. Reprinted from the *Archives of Medicine*, Vol. IX., No. 3, June, 1883.

A more physiological view of the action of remedies upon organic diseases, and a careful examination of clinical evidence would, it seems to me, prevent one from assuming the specific action of iodide of potassium in syphilis.*

As regards the general question, that of the specific action of remedies, I have not the time to present an argument to show its fallacy; and probably I could not do the matter justice. The belief in the specific action of drugs, *i.e.*, of the action of drugs against disease as such, is a comfortable belief to have; it apparently solves many of the problems of every-day medical practice. But many believe such a doctrine to be just as fallacious and unscientific as it is comfortable. I wholly agree with those who think this, and who believe that remedies act on the organism as a whole, or on its apparatuses, or on some of its tissues, or on its constituent chemical ingredients, in a *physiological* way, *i.e.*, by and through the operation of chemical and physiological laws already operative in the animal body.

In the second place, as to clinical evidence. This is the purpose of my paper; to place before you some cases which I think support the proposition that the iodide of potassium is efficacious, more or less, in non-syphilitic nervous diseases. In going through my case-books for the purpose of finding such illustrative cases, I have exercised great strictness, and as a result I have had but very few histories to read, and these I have condensed as much as possible.

My cases are nine in number, arranged in two groups.

In the first group are three cases of organic disease of the brain, in which many threatening symptoms were relieved, in some of them immediately and on different occasions, by the free use of the iodide. In all these cases post-mortem examinations were made, and the gross lesion found. In all of these there was no clinical or histological evidence of syphilis.

In the second group are six cases which are still living, some cured. I divide this group into two classes, *a* and *b*. The former is made up of three cases of organic cerebral disease in the adult, two of them cured, and the third twice relieved of most of his symptoms by the iodide. Class *b* is composed of three cases of basal meningitis with optic neuritis in little children, who recovered rapidly while using the same remedy. I attach much less importance to these infantile cases, because of the doubt there must remain as to their having been anything more than optic neuritis. Still they have a certain value in a purely clinical paper like this one.

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* For the opinion of various authors on these points see the end of the article.—[R. W. A.]

FIRST GROUP.

CASE I.—*Tumor of left crus cerebri.* G. W., æt. nine years. Seen 21st September, 1874. Had been a healthy boy. Parents and other children healthy.

In the month of April had measles without head symptoms. Early in May awkwardness of right side of body; gradually extending paresis from arm to leg; face unaffected. In August he walked like an old man, with his right shoulder drooping, right arm almost motionless; speech normal. During July and August had a great deal of occipital headache, relieved by cold. Since August, pain in various parts of head; more in front and behind. Of late the pain has been sharp, occurring in paroxysms, accompanied by nausea and vomiting. Irregular jerking movements of the paralyzed side, first noticed in August; none in the face. In the last ten days as above, but weaker; able to walk a little alone. Four weeks ago double vision, and since a squint; parents thinks vision is otherwise normal. The pulse has been observed by the family physician, Dr. Banks, to be habitually very slow, about 60, and at times irregular. Has had no convulsion or loss of consciousness. No recent injury to head, and never disease of the ear. Paroxysms of headache and vomiting often occur in the middle of the night.

On examination I found patient conscious, with right-sided hemiplegia; lower face affected. There was also palsy of the left sixth cranial nerve, producing convergent strabismus and diplopia. Slight rigidity in fingers of right hand. When patient attempts voluntary movements there is well-marked ataxia of the right upper extremity; no anæsthesia: pupils normal; vision impaired in left eye; opthalmoscope shows choked discs; on attempting to walk staggers very much; complains of vertigo; left side of body normal.

I advised the application of blisters behind the ears, and internally a saturated solution of iodide of potassium. On October 2d, patient being weaker, he, after vomiting in the morning, rather suddenly passed into coma. No convulsions. After forty hours regained consciousness with more paralysis on the right side, the same ataxia, and nearly complete loss of vision. Paroxysms of pain and vomiting never returned after this.

On October 4th, improvement began in right side and continued; mind clear.

On October 11th, is taking ten drops of the iodide solution three times a day; can move right leg.

October 20th, takes eighteen drops three times a day; more strength in arm and leg.

November 17th, takes forty-five drops three times a day; can raise himself up in bed. November 21st, taking sixty drops thrice a day; walks with some help.

Until December 8th this maximum dose of sixty drops was continued; after that date it was gradually reduced to forty drops on the 13th. Constant gain.

I saw G. on December 14th. He then walked about alone with a half-ataxic, half-choreic action of the right side. He presented a partial right hemiplegia, face and body, and complete palsy of the left sixth nerve; no anæsthesia. The optic nerves showed commencing white atrophy; no perception of light; no muscular atrophy; no headache.

My friend, Dr. J. C. Shaw, of Brooklyn, then took immediate charge of the case, though I saw it occasionally with him the first year. Iodide omitted in winter.

In 1875, from early in April to end of May, severe symptoms—headache, vomiting, cramps in calves of legs, and priapism—were relieved by the iodide increased slowly from .60 to 2.3 three times a day. After having been in bed for weeks, is again able to walk about with some right hemiparesis and hemichorea.

Dr. Shaw saw patient rarely after that until the spring of 1880, when after a series of anomalous symptoms he died. The autopsy made by Dr. Shaw revealed complete compression of the left crus cerebri and pressure on adjacent parts of the pons and cerebrum by a large irregular tumor.

The microscope showed it to be chiefly a sarcomatous growth with here and there large cells, either mother cells or modified ganglion cells.

CASE II.—*Cerebellar tumor—internal hydrocephalus.* Paul K., aged eight years. Seen in consultation with Dr. Malcolm McLean, of Harlem, on November 17, 1879.

In the past eight or nine months has suffered from diffused headache, attacks of vomiting, double exophthalmus, and staggering gait. Has been seen by many physicians, most of whom attributed the symptoms to "malaria." Child grew steadily worse in spite of treatment on this theory, and in August was taken to the Catskill Mountains. While there seemed worse; headache severe; staggered and vomited; was very weak. In September came under Dr. McLean's care, with above symptoms; no paralysis or impairment of intelligence. Parents stated that there have been no epileptiform seizures and no fever. Small doses of iodide of potassium caused improvement. Treatment suspended in October.

In last two or three weeks again worse; severe headache, much of it occipital and frontal. Great enlargement of the head and separation of sutures. Marked exophthalmus—staggering gait and pseudo-paraplegia. A few days ago there occurred sudden recession of the exophthalmus, and simultaneously there appeared a soft, fluctuating tumor or swelling in the right occipital region.

There is no history of injury to the head, or of causes of tuberculosis.

Examination.—Child pale but intelligent; speech normal; vision seems good by finger and color tests, but the ophthalmoscope shows double neuro-retinitis (choked disc) of moderate degree. No facial or head paralysis. Co-ordinates perfectly well. All the cranial sutures are wide open; anterior fontanelle closed; forehead not very prominent; no exophthalmus now. In the right occipital region, in the vicinity of the lambdoid suture, is a soft, compressible subcutaneous tumor, walnut size, whose contents beat synchronously with the pulse. The appearance of this swelling caused a relief to all symptoms except debility. It might be supposed that this swelling contained fluid derived from the hydrocephalus, but from its location I felt considerable doubt as to this.

Patient walked feebly in a staggering way; no paralysis or ataxia.

I made the diagnosis of internal cerebral hydrocephalus, probably from tumor of the cerebellum comprising aqueduct of Sylvius. I advised against puncture and aspiration of the newly formed sac, and recommended larger doses of potassium iodide.

Dr. McLean kindly wrote me December 30th of this year:

"We immediately increased the iodide of potassium from ordinary doses to from 1.5–2.4; so that he received amounts of the medicine varying from 6.–9. per day. The medicine never disturbed his stomach, and his symptoms were certainly ameliorated by the larger doses, which were continued for four months without interruption. The pains in the head were undoubtedly controlled by the medicine."

The child died in the early spring of 1880, and an autopsy by Dr. McLean showed a cerebellar tumor compressing the aquæductus Sylvii and the venæ Galeni, thus causing ventricular dropsy. Tumor was fibro-sarcoma.

It was well that the externally presenting sac was not punctured, for it turned out to be the extruded lateral sinus.

CASE III.—*Tumor of the cerebellum.* J. J., aged 14 years. Seen first on July 29, 1880. Had been a healthy boy. At three years had whooping-cough severely with several convulsions. Parents deny convulsions or petit-mal since.

About January 1, 1876, J. fell heavily on a stone walk, striking his head so hard as to make him unconscious; did not vomit. In April of that year he began to have curious vomiting spells in the early morning, followed by violent occipital headache. The patient describes the vomiting as not preceded by nausea, and the rejected matters contained no food. After having had these attacks for several days, one afternoon J. fell unconscious and had a general convulsion, repeated in the night. After this J. carried his head inclined to the left shoulder, his occipital headache continued, and he had a stiffish feeling in the neck. The vomiting did not return, and there was no delirium.

At the end of May he had gradually become paralyzed generally, but more on the left side. He had pain in his eyes, with rapid failure of sight. Drs. Agnew and Knapp found white atrophy of the optic nerves. No recovery of sight since. (It is very probable that during April there had been choked discs, with fairly preserved vision.) Speech was never affected.

Spontaneous improvement occurred, and in July, J. was able to sit up, and gained rapidly in all respects except sight. Some disability in use of hands and walking remained. He grew well, and was taught at the school for the blind. Has been very intelligent. No special symptoms occurred for nearly four years, viz.: until May of this year (1880) when he began to have attacks of occipital pain and vomiting; occasionally had pain in left mastoid region, and numbness in left side of chin, and around left corner of mouth. A few days ago was found unconscious; probably had had a convulsion. Admits occasional dizzy or unconscious spells of momentary duration. Is still able to be up all day, dressed.

Examination (July 29th).—Eyes in left conjugate deviation; sightless; pupils wide; nerves bluish white. Tongue straight; right hand, 20°; left, 25°. Left leg stronger than right. Consequently has right hemiparesis; no tendon reflex at knees; walk is staggering, more off toward his left. There is no distinct ataxia, and the walk is not of the type called cerebellar; no anæsthesia. I gave him a mixture of bromide and iodide of potassium, of each salt about 1. at night,; quinine, sherry wine, and food.

Sept. 29th.—Patient improved wonderfully in first month of above treatment. Early in September had a sort of convulsion, and since more or less occipital pain; objective symptoms as above.

Nov. 14th.—Poorly of late. Occasional attacks of occipital pain and vomiting (without nausea); rather frequent attacks of petit-mal, or perhaps more strictly speaking syncopal attacks, usually associated with headache. In last twenty-fours hours has been semi-comatose, at times vomiting. Pulse weak. Ordered ext. digit. fld., .06, and tr. opii .18 by mouth.

Nov. 16th.—To-day better, and is ordered ten drops of a saturated solution (equal parts) of iodide of potassium three times a day, to be increased each day by two drops at a dose. The small dose of bromide heretofore given (about 1.) stopped.

Dec. 6th.—The iodide has been gradually increased to forty drops three times a day, with the best results; no headache or vomiting or syncope since beginning iodide. No bromide. Rich food and sherry.

Examination shows a new symptom, viz., occasional twitching and distinct ataxia of the right upper extremity; none in the legs; perhaps a trace of ataxia in left hand. Absolutely no tendon-reflex at knees. Right hemiparesis; no anæsthesia: face not paralyzed. Is up all day, and walks out of doors occasionally. Iodide to be gradually reduced.

Several times during the winter and spring of 1881, J. had a return of occipital pain and syncopal attacks; more recently of cervical pain also. These attacks were invariably cut short by blistering the nape of the neck or the mastoids, and by giving at once the full doses of iodide of potassium, viz., from forty to fifty drops three times a day. Previous to December 21st the blisters had not been used, so that we may conclude that the more potent agent, in affording relief to the very distressing and threatening symptoms was the iodide of potassium. The relief usually appeared in two or three days. Between the exacerbations the dose of iodide was from ten to twenty drops, and he had a variety of tonics.

The summer of 1881 was exceptionably favorable for J. He was very well and happy. Though blind and slightly ataxic he enjoyed life, and was very cheerful. He had learned to do many delicate manipulations with his hands.

Oct. 12th.—J. was seized with convulsions, vomiting, and a gradually increasing pyrexia. Died comatose on 14th at midnight, with axillary temperature of 39.5° C.

Autopsy showed a tumor involving a large part of the inferior portion of the right hemisphere of the cerebellum, forcibly compressing the underlying portion of the mesocephale. The upper three-fourths of the same right hemisphere of the cerebellum was occupied by a cyst containing a clear fluid. The bottom of this cyst is the solid tumor referred to above. The cyst has disintegrated the upper and middle portions of the vermis superior.

The cerebral convexity showed abundant heavy patches of purulent subarachnoid meningitis, chiefly along vessels. The microscope showed, in fresh serum preparations, tubercle-like masses round about vessels, and at their bifurcation. This meningitis was the cause of death.

A microscopic examination of the solid cerebellar tumor showed the subcystic tumor to be mainly sarcomatous, cellular and vascular, with foci of amyloid degeneration.

The family are all unusually healthy. Besides J. there are seven living children who are pictures of health. The father and mother are perfectly well, and always have been. The teeth of patient were normal, and he was a well-developed lad of rather hydrocephalic aspect. No suspicion of specific disease could be entertained in this case.

SECOND GROUP, CLASS A.

CASE I.—*Left hemi-paræsthesia cured by potassium iodide.* Dr. J. K., U. S. A. æt. forty-five years, seen December 8, 1877. Had always enjoyed good health. While on duty in a Western State, December 13, 1874, had a sudden attack of left hemi-numbness—face and body. There was only very slight loss of motor power, if any. The sensation, which for a long time was intense, was a mixture of hyperæsthesia and numbness. Special senses unaffected. The paræsthesia has diminished in extent and intensity, but is still constantly present in the ulnar side of the left hand and outer side of left leg; occasionally in left cheek.

Examination shows no actual paralysis, though there is awkwardness of the numb parts. There is no true anæsthesia, and neither ataxia nor chorea. The heart is large and beats heavily; no murmur (?). Has had several angina-like attacks. Patient adds, that when first attacked in 1874, his left external rectus was paralyzed for two weeks. There is well-marked dementia, slowness of intellect, and loss of memory. Patient has failed in his examination for promotion, in spite of hard work.

Habits always good; positively and repeatedly denies syphilitic infection, or any symptoms. Notwithstanding my faith in the doctor's denial, I gave him iodide of potassium freely, and in a few weeks all the paræsthesia disappeared. Subsequently the case developed into one of dementia paralytica, with occasional epileptiform and apoplectiform attacks. He still lives in a subjectively happy imbecile state.

I regret that the notes of the case contain no record of the doses of iodide employed. The relief was striking, however.

CASE II.—*Paralysis of third cerebral nerves; paresis and ataxia of limbs; relief* *in two attacks by iodide of potassium.* W. R. B., æt. twenty-nine years. Referred by Dr. Agnew, July 16, 1878.

Former health poor. Tobacco and beer used to excess; intense dyspepsia, with cardiac disorder and abdominal paræsthesia; catarrh of bladder; impaired memory. Positively denies syphilis.

July 2d.—While camping in the woods, awoke with paresis of left third nerve (ptosis and diplopia), which rapidly became complete paralysis. No other symptoms. Galvanism and iodide were employed, and strychnia hypodermically.

July 20th.—In last two or three days a numbish feeling began in feet and has extended to middle of the thighs; legs weak; aching pains from sacrum to feet. At times finger tips are also numb. No vesical weakness. No patellar tendon-reflex. Was ordered, Squibb's fluid extract of ergot, dry cups to spinal region, and rest in bed. Optic nerves normal.

About the beginning of August the right third nerve also became paretic, and then paralytic symptoms in legs and arms were the same ; they were all signs indicating a lesion involving the crura cerebri. Ergot was continued, and iodide of potassium added to the treatment.

August 27th.—Very much improved; walks quite well; arms seem weak to patient, but he can squeeze 59° with right and 53° with left hand. Both third nerves better; images nearer together; less dizziness. Has been taking more iodide and less ergot of late. Uses galvanic current to hands and eyes. Ordered: cease ergot and increase iodide from present dose of forty-five to sixty drops three times a day.

31st.—Improving. Right internal rectus nearly normal; can open left eye better. At times a trifle of numbness in outer part of feet. In spite of sixty drops of solution of iodide three times a day his digestion is better than for a long time. To continue iodide, and to take 4. of dialyzed iron at bedtime.

September 7th.—Right eye moves normally; left nearly well; internus and levator palpebræ are weak. Ordered: Continue sixty drops of the solution of the iodide *t. i. d.* [also other remedies for nervousness].

12th.—Goes to the country nearly well; left third nerve still paretic. Iodide to be decreased by two drops each day. Galvanic application to be kept up to the eye. Takes also quinine, iron, digitalis.

23d.—Advised by letter to resume sixty drops of solution of the iodide.

December 11th.—Patient was *in statu quo ;* free from paralytic symptoms except in distribution of left third nerve. Is now taking thirty drops three times a day. Also bichloride of mercury .004, in elixir of calisaya, three times a day.

During the spring and summer of 1879, the left third nerve varied in its condition, and the dosage of iodide was varied accordingly, ranging from twenty to sixty drops *t. i. d.* He had it most of the time, and it never produced any disagreeable effect. There was no return of paretic symptoms in the limbs.

Nov. 7, 1879, Mr. B. again came under observation. The left eye was as before, and he had some new symptoms. Slight numbness in the deep and superficial branches of the left trigeminus (including tongue). Two or three weeks ago the legs were weak, and an approach to numbness was observed in the thighs.

Examination showed paresis of left third nerve; when right eye is kept closed, and a strong effort of the will made, the left lid can be raised, and even the internus contracts (slow conduction in nerve). In accommodation with convergence the left pupil contracts like the right. Left facial muscles are perhaps weak; the æsthesiometer shows some anæsthesia in distribution of the left trigeminus; pricking is well felt. Tongue projects straight. Hands not anæsthetic or numb; grasp, R., 69° and 64°; L., 57° and 56°. Knee reflex entirely absent. Stands well with eyes closed. Sexual debility; optic nerves normal; denies fulgurating pains, and again denies syphilitic infection or symptoms. Last winter his wife was delivered of a very healthy baby. Ordered: Increase iodide solution from present dose of thirty drops *t. i. d.* by 5 drops each day, to 120 drops *t. i. d.*, then to decrease. Also ordered phosphide of zinc, .006; ext. nucis vomicæ, .02; quiniæ sulph., .10; in a pill *t. i. d.* Goes home to Central New York.

Oct. 2d, 1880. Returns to New York. Mr. B. carried out the above treatment faithfully for a long time, and was entirely relieved of all symptoms, except sluggishness of the left third nerve. He remained fairly well until June of this year. Then, after resuming the free use of beer and tobacco (denied sexual excess), he noticed awkwardness and numbness of the hands, staggering in walking, and legs seemed weak. No change in left eye. Return this autumn of numbness in left supra-orbital region. No headache.

Examination showed a titubating and coarsely ataxic walk; staggering when standing with eyes closed; no patellar reflex; marked anæsthesia and ataxia of both hands, especially of right. Left eye as described above (imperfect and slow conduction in third nerve). Pupils active; optic nerves normal. The ataxia of hands in test with closed eyes is typical. Slight anæsthesia of left forehead.

The patient remained under treatment until April, 1881, when he went home without improvement; really more ataxic and anæsthetic in hands and feet.

Throughout the long duration of the case, there was no headache (only paræsthesia at vertex when anæmic in first winter of treatment), and the optic nerves have escaped injury. All the symptoms point to disease of the left crus cerebri, extending over toward the right, and the case bears a certain resemblance to Cases I. and III. of the first group, in which tumors were found, except that the patients had blindness from disease of the optic nerve.

I should add that in the last visit of the patient to town, from October, 1880, to April, 1881, persevering attempts at treatment were made. He had the iodide solution carried up to 175 drops three times a day; bichloride of mercury in moderate doses; strychina; the actual cautery behind ears and down the spine, and galvanism. The disease made slow but sure progress all the time. Yet, twice before, in 1878 and in 1879, the iodide had, in the most evident manner, removed all symptoms except the paresis of the third nerve on the left side.

My friend, Dr. Charles McBurney, who is known to many of you, had been well acquainted with Mr. B., and was disposed to place reliance in his denial of syphilis.

CASE III.—*Right hemi-epilepsy and aphasia cured by iodide of potassium.* Mr. J. L. C., æt. twenty-four (?), was seen September 30, 1879. This gentleman was brought from Newport by Dr. George Engs, and was placed under my care by his family. When I first saw him he was conscious, but suffering from frequently repeated epileptiform attacks in the right face. From a variety of sources the following history of the case was gradually obtained. Patient is a large, well-developed young man, who has always enjoyed good health. For more than a year Mr. C. has worried about some secret trouble, and has become dull, complained of insomnia, has lost his interest in reading and in music, of which he was very fond. Last winter he contracted a severe bronchitis, which has very slowly passed away. No injury to head; positively denies syphilis, and bears no sign of it.

About the last of August he came in one day from the beach at Newport, and said he had had a sunstroke. No one was with him at the time, so that the nature of the attack remains a mystery. It could hardly have been a sunstroke, as there were no others in Newport that day, and the heat was not excessive. Judged by the light of subsequent events, it must have been a first epileptiform attack.

Soon thereafter Mr. C. went to Lenox, Mass., and led an active social life, dancing, playing lawn-tennis, etc. Complained of headache on the way to Lenox, and while there. While there had three or more epileptiform seizures, varying from a "faint" to an attack in which the right arm was stiff and unmanageable. This decided spasm, witnessed by an intelligent layman, occurred about September 15th. The next day he went alone to Boston, turned up at a friend's club greatly confused, asking who he was, and writing his name on a card for use in case of trouble; came to Newport in a couple of days, and was there under Dr. Engs' observation. When seen on September — was confused; used wrong words, or rather had to struggle to find or enunciate the right word (aphasia).

On September 23d, in Dr. Engs' presence, after struggling to find a word, he was seized with a full epileptic fit, probably stronger on right side. He bit his tongue slightly. Pulse slow; no fever; no albumen in the urine. September 24th, epileptic attack in evening. Dr. S. Weir Mitchell saw patient; found optic nerves normal, and would make no diagnosis. Was given bromide of potassium in doses of 1.2, frequently repeated. On 25th had three or four attacks, chiefly affecting right face, arm and leg. Since he has had innumerable partial attacks every day, affecting the right face and arm; not always with loss of consciousness. For example, on September 28th he had at least twenty-five seizures; more frequent in last forty-eight hours. In the last day or two the spasm has tended to restrict itself to the face on right side, and has not been accompanied by insensibility. During the week there has been progressive abolition of speech; now says but two or three words. Has had from 8. to 10. of bromide of potassium daily in last three to four days.

Description of facial hemi-spasm, as observed frequently on September 30th and on October 1st: "First there is a tonic spasm of superficial muscles, especially the buccinator and levator anguli oris; the mouth is strongly drawn to the right; eyes closed; at same time jaws are motionless, sometimes closed, sometimes opened about thirty mm. In a few seconds clonic movements appear in superficial facial muscles, a few chewing movements are made, and a stream of saliva flows, partly caught upon a cloth held by patient, who is perfectly conscious. Some saliva runs back into the larynx and causes cough. Pupils remain normal. A few times the cervical muscles on right side seemed stiffened; tongue not bitten, but a right canine tooth has caused ulceration of inside of lip." These attacks were the residua of the previous hemi-spasms, and of the still older general spasm. They presented all the characters of the Jacksonian or motorial epilepsy, which is so certain an indication of a gross local cerebral lesion.

Oct. 1st.—In the night Mr. C. had from thirty to forty attacks of mixed facial and trigeminal spasms on the right side. Arose, and turned down the gas, saying distinctly, "Down, down." Liquid food causes strangling and coughing. Has gone to water closet himself. Axillary temperature normal and pulse 90. Last evening was cupped behind ears, and had chloral besides bromide: .30 of chloral and 1.20 of bromide every four hours. At noon said "beef tea"; looks dull and sleepy, great drewling, tongue protrudes to the right; toward evening fewer spasms; pronounces his name on demand; mind clear; small blisters behind each ear; no evident paralysis of face, arm, or leg, but aphasia and agraphia are practically complete. Patient has the vacant, helpless, impatient look of aphasics when asked a question. Pulse 100.

Oct. 2d.—Very much better. Slept a great deal, and had few spasms in face last night; drinks more easily; sits naturally on lounge and shakes hands; with slowness says about a dozen words; no headache; recalls names of Drs. Engs and Mitchell. For the first time in forty-eight hours no spasm occurs during my visit; axillary temperature 36.4° C.; pulse 102. Less drewling; tongue still goes to right; order 20 drops of a saturated solution of iodide of potassium, .30 of chloral, and 3. of bromide at one dose, three times a day. The optic nerves were examined in the first few days of the treatment and afterward, but found healthy.

It is unnecessary to continue a journal of this case. The above mixture was continued for several weeks, the bromide being reduced to 2. and the iodide increased to 80 drops, three times a day. After October 17th no chloral was given. The iodide was further increased, a maximum being reached on October 26th, when 120 drops were given, with 2. of bromide three times a day.

Not many days later the bromide was omitted and the iodide given with a tonic in doses ranging from 100 to 50 drops three times a day.

At no time did the iodide produce any unpleasant effects. Toward the end of October some bromism appeared.

The improvement was steady in all these four weeks; the local spasms became fewer, and ceased before the end of the month. Speech steadily increased, and toward the end of the month writing was begun.

Mr. C. came to see me about getting married last winter. I again questioned him about syphilis, and he gave me his word of honor that he had never had a venereal sore or a suspicious symptom.

He has remained perfectly well.

SECOND GROUP, CLASS B.

Cases of double optic neuritis, probably due to basal meningitis, in children; apparent good results from large doses of iodide of potassium.*

CASE I.—A little girl, aged six years, was brought to my class at the Manhattan Hospital a couple of weeks ago, with the following simple history; For two or three weeks she had complained of headache, had vomited frequently, and on February 9th (a week ago) internal strabismus appeared. The patient has not complained of impairment of vision; she has not had fever, spasm, or delirium. Constipation has, however, been marked. She is anæmic looking; a small brother of hers probably has phthisis, and one child of the same parents is said to have died of "brain fever." My assistant at the Manhattan Hospital, Dr. Adam, immediately examined the child's eyes with the ophthalmoscope, and found double neuro-retinitis; a diagnosis which I concurred in, and which was verified by Dr. Webster in the Ophthalmic Department of the hospital. Consequently, the most important symptom was the one revealed to us by the use of the ophthalmoscope. I made the diagnosis of basal meningitis localized about the chiasm of the optic nerves, probably without tubercular deposit. The child was blistered behind the ears, and given .60 of potassium iodide three times a day, with instruction to increase the dose by .30 per dose, every second day.

The child now does not seem sick, and were it not for the convergent squint, one would probably consider her as only a delicate anæmic child. In the last few days, the headache and vomiting have ceased, and improvement has begun.

CASE II.—Referred for examination to Prof. H. Knapp, on May 2, 1877, a girl, aged four years, previously healthy. First symptoms noticed about five weeks before examination, consisting chiefly in dullness, irritability, slight headache, and, on one occasion only, vomiting. Two weeks later internal strabismus (one eye) suddenly set in, and has persisted. No fever, spasm, or delirium. Previous to this attack there had been no emaciation, or cough, or ill-health of any kind. Dr. Knapp found double neuro-retinitis, with paresis of external rectus of one eye. On examination, I found the child with the above optic symptoms, and very cross; the buccal temperature was 37.25° C., and the pulse 96, perfectly regular. I made the diagnosis of non-tubercular localized basal meningitis, and expressed the opinion that the child's life was in no danger, though vision might remain considerably impaired. Dr. Knapp was giving potassium iodide, which I also advised. A few days ago Dr. Knapp informed me that a few weeks after I saw the child the strabismus disappeared, and that the neuro-retinitis gradually gave place to atrophy of the optic nerves, which, fortunately, was but slight, so that vision is now nearly perfect.

CASE III.—A little girl aged five years was sent to me for examination by

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* From a clinical lecture delivered at the College of Physicians and Surgeons, New York, Saturday, February 23, 1878.

Prof. C. R. Agnew, February 14, 1878. I learned that the child had passed through an attack of chicken-pox early in January, without fever or apparent ill-health. About January 19th, the left eye "turned in," and strabismus has been constantly present since. No other symptoms have been observed—no fever, headache, irritability, etc. The mother states that one of her former children, at the age of eleven months, had convulsions and fever, became unconscious, and died in two weeks.

Examination of the eyes by Dr. Agnew reveals "double optic neuritis, with some stuffing of the disks; hypermetropia, 5. of the disk."

I made the same diagnosis as in the case sent me by Dr. Knapp (Case II.); viz., local basal meningitis of a non-tubercular nature. I advised blisters behind the ears, and large doses of potassium iodide. The case did very well.

I submit this contribution, well aware of several objections to the thesis it supports.

It may be said that the improvement observed was the result of the so-called *vis medicatrix naturæ*, a spontaneous improvement. This might more especially be said with reference to the cases of infantile basal meningitis and optic neuritis. Case I. of the first group is likewise open to this objection, but in the other cases it is different. In Case II. relief was twice obtained in an evident manner from the iodide; in Case III. threatening symptoms repeatedly passed away within a few hours after giving full doses of the drug.

The first class of the second group is more demonstrative of the curative powers of the iodide. In Case I. we see hemi-paræsthesia of three years' standing disappearing in a few weeks; in Case II. symptoms quite positively indicative of organic disease of the crura cerebri twice relieved (all but slight weakness of the third cerebral nerve) by the iodide; and in Case III. the symptom-group which we often call Jacksonian hemi-epilepsy, with aphasia, was completely and permanently cured. My experience with these hemi-symptoms is, that they nearly always indicate a tumor in the brain, and I have seldom seen them cured.

To the cured cases it may be objected that the patients consciously or unconsciously deceived me as to the existence of syphilis. It so happens that the subjects of the class just referred to were men who by their temperament and their relations to me would have been very unlikely to deceive, and they all three were told how essential it was to a proper treatment of the case that they should tell the exact truth.

But there are other tests, besides a belief in the truthfulness of the patients. They bore no external evidences of syphilis in the shape of scars or enlarged glands. Case II. demonstrated the non-syphilitic nature of its lesion by a third relapse with new symptoms utterly resisting the iodide. Case III. has remained well for two years without treatment.

Case I. had symptoms of failing mind when he consulted me, and has since been a good example of paralytic dementia without positively exalted notions. The non-syphilitic nature of this dementia has been shown by its very slow development, and by the fact that one or two trials of mercury and iodide of potassium proved unfavorable. Besides, the patient, as a physician and an army man, was the least likely of all to deceive me as to the origin of his trouble.

In my first experience with these cases, I gave the iodide of potassium in medium doses, from thirty to fifty drops of a saturated solution three times a day, but since 1877–8 I have used much larger doses and with no unpleasant results.

It is surprising how well patients of all ages bear doses of from fifty to one hundred and fifty drops of the solution without iodism or gastric catarrh. I give it largely diluted, in from one-half to a full tumbler of water, and always on an empty stomach, to diminish the risk of decomposition. In the last two or three years I have adopted a plan which I think further assists immediate absorption of the iodide as such, viz., the use of Vichy instead of common water as a vehicle; or, as a substitute for poor patients, a solution of bicarbonate of sodium. I might add that in several patients, including one of those referred to in this paper (Case II. of second group), digestion has been improved by the iodide given in this way.

Even if the iodide of potassium cannot cure organic diseases of the brain it seems to relieve symptoms. If by the free use of such a remedy, one not directly harmful, we can diminish intracranial tension, remove œdema, or perhaps check the growth of some neoplasm, thereby relieving pain and other distressing symptoms, would not this be a gain to our therapeutics?

IDEAS OF DIFFERENT AUTHORS BEARING ON THE SUBJECT OF THE FOREGOING PAPER.

BARTHOLOW, R., A Practical Treatise on Materia Medica and Therapeutics, 3d edition, 1880, p. 191, says: "But few affections of the brain, non-specific in origin, are benefited by the iodides."

Bartholow, R., A Treatise on the Practice of Medicine for the Use of Students and Practitioners, 3d edition, 1882, p. 568, in speaking of the treatment of intra-cranial tumors, says: "There are two remedies which ought always to be used—iodide of potassium and ergot; for although only syphilitic and possibly aneurismal tumors are remediable, the case under treatment may be one of them."

Edes, Robert T., Therapeutic Hand-book of the United States Pharmacopœia, etc., 1883, p. 255, says: "The curability of any disease by iodide of potassium, however, does not warrant a diagnosis of syphilis." Page 256: "It should be given freely in all cases of cerebral tumors, and often in meningitis."

Flint, Austin, A Treatise on the Principles and Practice of Medicine, etc., 5th edition, 1881, p. 726, when speaking of the treatment of tumors within the cranium, says: "It is, however, claimed, that certain remedies, namely, the iodide of potassium, the bichloride of mercury, and arsenic do have such an influence in such cases" (meaning non-syphilitic growths).

Hamilton, Allan McLane, Nervous Diseases; their Description and Treatment, 1878, p. 202, under the treatment of tumors of the brain, says: "It has been my practice in every case to place the patient upon an anti-syphilitic course of treatment."

Hammond, William A., A Treatise on the Diseases of the Nervous System, 7th edition, 1881, p. 324, under the head of treatment of tumors of the brain, says: "So far, however, as other* tumors of the brain are concerned, there is no treatment calculated to cure the patient, unless a syphilitic taint can be ascertained to exist. It is well, however, even when there are no positive indications of the presence of such a diathesis, to act upon the presumption that it does exist, and to administer mercury in some form with the iodide of potassium."

Obernier, F., in Ziemssen's Cyclopædia of the Practice of Medicine, vol. xii., Diseases of the Brain and its Membranes, when giving the treatment of tumors of the brain and its membranes, p. 288, says: "A trial of it† should not be neglected."

Ross, James, A Treatise on the Diseases of the Nervous System, 1881, vol. ii., p. 567, when giving the treatment of focal diseases of the brain, says: "With the view of promoting absorption of the morbid growth, iodide of potassium has been administered in large doses and with apparent benefit."

Stillé and Maisch, The National Dispensatory, 2d edition, 1879, p. 1161, under the subject of potassium iodide, say: "In many cases of *paralysis*, due, probably, to pressure upon a motor centre, or upon a nervous trunk, produced by syphilitic or other swellings, the medicine is often singularly efficient and should never be omitted from the treatment."

Wilks, Samuel, Lectures on Diseases of the Nervous System, 1878, p. 461, in general remarks on remedies, says: "In cases of epilepsy and many obscure nervous affections, I usually commence with this class of remedies,‡ knowing that a curable disease has sometimes ended fatally because they have been overlooked."—[R. W. A.]

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* Meaning other than aneurismal.

† Meaning iodide of potassium.

‡ Meaning iodide of potassium and perchloride of mercury.

ON THE EFFICIENT DOSAGE OF CERTAIN REMEDIES USED IN THE TREATMENT OF NERVOUS DISEASES.*

MR. PRESIDENT AND GENTLEMEN: I have been led to prepare this paper by the following consideration. I frequently see uncured cases of nervous disease for which the attending physician has prescribed the proper remedy, but having exhibited it in doses which, though justified by medical authorities, were wholly insufficient to influence the disease, he has failed. This has been more especially true of chorea, of cerebral and spinal syphilis, of certain neuralgias. In these cases the physician had been wanting in the experience and in the courage necessary to fight his way through opposing tradition and book-authority to success.

There are several evident causes for this timidity, which in a negative way is nearly as injurious to the patient as too great rashness would be.

In the first place, the influence of teachers in medical schools and of writers of text-books is thrown in favor of small or medium doses. Few if any teachers or writers take special pains to indicate the maximal doses of potent drugs; they teach in a condensed form, and with an eye to the safe training of students. This is very well as applied to students, but a time comes when a physician in active practice wants to know just how much physiological effect he can obtain with certain remedies without positively endangering his patients' lives. In the present state of our medical literature, unless he have time and opportunity to hunt through the files of the leading medical journals for detailed observations, or to read monographs on experimental therapeutics, he must work out his maximal doses for himself at the cost of much time, of some anxiety, and of not a few failures.

It seems to me that works on therapeutics intended for the practitioner should give, for each important, physiologically active remedy, a paragraph on maximal doses, clearly indicating the amounts necessary to produce the physiological effects (on man), which are often inseparable from remedial effect. These data should be taken from monographs and special articles on the subjects by men who have had experience in the use of the drugs mentioned. For I take it as granted that it is now just as impossible for one man to give us a satisfactory, practical work on therapeutics, as it is for one to produce a uniformly excellent work on the practice of medicine.

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* Read before the Medical Society of the State of New York, Feb. 7, 1882. Reprinted from the *Archives of Medicine*, vol. vii., No. 2, April, 1882.

In the second place, I have observed that many capable druggists are alarmed at doses of certain remedies which are not only harmless, but essential to success. I clearly remember that, when a student, I heard the late Prof. Freeman J. Bumstead relate, with a mixture of amusement and anger, how a leading druggist had sent to him to inquire if he really meant to give 6. of bromide of potassium at one dose. This was twenty years ago. Yet, only a few days since, a patient told me that her druggist told her that she must have a very strong stomach to stand such powerful medicine (she was taking .004 of biniodide of mercury and 3. of iodide of potassium three times a day, and under this in one week had lost nearly all her syphilitic pain). Very frequently have I had prescriptions for my usual doses of Squibb's conium returned for revision by the careful pharmacist. I intend nothing derogatory by these remarks, for druggists are supposed to know only the maximal doses of remedies as given by books, and they but do their duty in sending a prescription back for revision, if anything in it seems wrong; for my part, acknowledging a liability to error, I am always glad to see this healthy doubt applied occasionally to my prescriptions; yet I would not have physicians allow themselves to be influenced by the remarks or practice of druggists. Philosophically, the two professions are absolutely separated; the one furnishes the other with the proper implements of treatment in the best possible condition; and it is the function of the physician to determine by scientific knowledge and by experience how, when, and how much these implements shall be employed. In more senses than one the physician is responsible for the dosage of remedies.

In the third place, it has seemed to me that our large manufacturing drug-firms exert a baneful influence upon therapeutics. They have flooded the country with formulas and ready-made compounds, and thus relieved the physician of the necessity of exerting his power to extemporaneously devise the compound required for the individual patient before him. Increasing numbers of physicians, instead of adapting the materia medica to their patients, practically adapt their patients to an already prepared stock of elixirs, pills, and mixtures. It is so convenient to order one of these, so much easier than to weigh the indications presented by the case, to estimate the patient's susceptibility, and then to write out a good prescription for the case, or more exactly speaking, for the patient.

I propose to review briefly the posology of a few drugs—giving the doses as stated by the best authorities, by writers on therapeutics, and by clinicians, and then stating the doses which I believe to be useful and safe.

I wish it particularly understood that in advocating larger doses of these remedies I do so only on the basis of a tolerably large experience, and not at all from any theoretical scientific considerations. At the same time that I advocate efficient doses, I am carefully observant of all the circumstances which render patients susceptible, and always make an allowance for idiosyncrasy. Thus, in first prescribing a potent remedy, I take into consideration the age, sex, and size of the patient; and also make an estimate of his general condition, and note particularly the state of his circulatory organs. Then, for a patient whom I see for the first time, I order very small doses, doses such as the books justify, and by steady increase feel my way, fearlessly because watchfully, to the larger doses, often seemingly dangerous doses, which really affect the organism and may cure the disease.

In this matter I make no claim to originality, and would not affirm that the doses I recommend are always essential to success; I simply sum up my experience and place my results at your service.

I.—FLUID EXTRACT OF CONIUM.

(*Extractum conii fructûs fluidum.*—U. S. P.)

Doses as given by authorities on therapeutics and materia medica:

Is not mentioned by STILLÉ and MAISH, by STILLÉ, by NOTHNAGEL, nor by GUBLER.

WOOD. Therapeutics (1880), p. 371. Dose, .06–.10.

BARTHOLOW. Materia Medica (1880), p. 409. Dose, .12–.30, increased to 2.4.

RICE. Posological Tables (1879), p. 28. Dose, from .18–.30, to be increased with caution.

Doses as given by clinicians:

Conium, in the form of fluid extract, is not, to my knowledge, mentioned by any standard writer on the practice of medicine.

MEIGS and PEPPER, Diseases of Children (1870), p. 565, article chorea, refer to Dr. J. Harley's doses of succus conii with apparent astonishment.

To Dr. JOHN HARLEY (The Old Vegetable Neurotics, London, 1867) we owe the present rational or physiological use of conium. He swept away the former traditions of the potency of the drug, and showed that most of its preparations were inert. He obtained definite physiological and therapeutical results from the succus conii, administered in doses of from 8. to 32. By means of these quantities he obtained the paresis of third nerves, arms and legs, which is the characteristic result of conium action on the spinal cord.

The prototype of our excellent officinal preparation, the fluid extract made by Dr. Squibb, was unknown to Dr. Harley until just as his book was going to the press (p. 94, note).

Dr. Squibb and Dr. Manlius Smith had, however, read a paper before this Society, at its meeting in 1867, entitled: "An attempt to answer the question, Which part of conium is the best for medicinal use?" (See Transactions of the New York State Medical Society for 1867.)

Ever since, we, on this side of the Atlantic, have possessed by far the most reliable and the most powerful preparation of conium; but I am sorry to add that it has been used rather inefficiently, and that even intelligent physicians are afraid to use the only doses which have any effect.

I have used conium a good deal in the last ten years, and have always employed the fluid extract as made by Squibb. I have tried it in chorea, in spasm of paralyzed limbs, in general irritability, and in insomnia.

When the indication is present, as in chorea, to obtain muscular relaxation, after a few tentative doses of 1.2–2.4, I give at one dose 4.–6. cc. These doses cause drooping of the upper lids (sometimes diplopia) and paresis of the arms and legs. I do not repeat the dose until after all the effects have passed off —in from 12 to 24 hours.

In a case of chronic, adult chorea of 14 years' standing, which I almost perfectly cured in 1872–3, at the Epileptic and Paralytic Hospital on Blackwell's Island, a large part of the result (a very remarkable result in my experience) was attributable to paresis daily produced by a teaspoonful of Squibb's extract of conium for a month or more.

Many cases of insomnia, with wakefulness in the first part of the night, more especially those with fidgets or physical restlessness, are very much benefited by conium. I usually give 1.2 cc., with 1.2 of bromide of sodium in camphor water, at bedtime, to be repeated if necessary. In some cases (male adults) I give 3.–6. cc., at one dose in the mixture, not to be repeated. Such a sleeping-draught prescription has been repeatedly returned to me by druggists, because they thought the dose enormous. Indeed, I usually warn patients that the druggist may comment on the dose.

If we have a clear indication to give conium, we ought to give enough to fulfill the indications, and this cannot be done without obtaining the physiological effects. With due precaution, there is a wide and sure distance between physiological and toxic effects, yet, with reference to remedies such as I shall refer to, how few physicians understand and appreciate that the curative effects are obtained in just that interval between physiological and toxic effects. To be successful we must be bold, as bold as physiological knowledge can make us, and yet as cautious in the first giving of powerful drugs to a patient as if we had no courage at all.

ON THE EFFICIENT DOSAGE OF CERTAIN REMEDIES USED IN THE TREATMENT OF NERVOUS DISEASES.*

II.—CRYSTALLIZED ACONITIA OF DUQUESNEL.

(*Aconitia.*—U. S. P.)

Doses as given by authorities on materia medica and therapeutics:

STILLÉ and MAISCH, National Dispensatory (1879), p. 101. Primary dose .00024 two or three times a day. It is recommended in doses of .0005.

WOOD, Therapeutics (1880), p. 180, makes the truly astonishing statement that: "The alkaloid is officinal, but, on account of its intense activity, should not be given internally."

This was printed more than a year after the publication of the New York Therapeutical Society's report on aconitia in the *New York Medical Journal* for 1878. See also p. 367.

BARTHOLOW, Materia Medica (1880), p. 44, simply quotes the New York Therapeutical Society's formula. No personal statement as to doses.

RICE. Posological Tables (1879), p. 5: "Aconitia; aconitine. Alkaloid from aconite. The commercial product is an impure mixture of alkaloids. The dose is .0004 to .0005 increased with caution. Chiefly externally."

NOTHNAGEL and ROSSBACH. Arzneimittellehre (1878), p. 721. Aconitia is little employed internally. Dose, 0.004, and the daily quantity as 0.03.

This cannot refer to Duquesnel's aconitia. It might be a safe guide for giving Merck's aconitia, which is very impure and of doubtful efficacy.

GUBLER. Leçons de Thérapeutique (1877), pp. 147, 8. Prof. Gubler may be considered as the introducer of Duquesnel's aconitia. In articles, besides in this book, he was the first to indicate its wonderful efficacy in neuralgia, particularly trigeminal neuralgia.

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* This article is a continuation of one which appeared with the same title in the April number of this journal, page 177.—Reprinted from the *Archives of Medicine*, vol. vii., No. 3, June, 1882.

He recommends 0.0005, or less at first; gradually increased to 0.002–0.004–0.005.

Doses recommended by clinicians:

As Duquesnel's aconitia has been known so few years, and has been in use less than four years in this country, it is not singular that our principal text-books do not speak of it. Still one is surprised to find that Prof. Flint in the last edition of his "Practice," dated 1881, does not refer to aconitia among the remedies which may cure neuralgia.

Hammond, Diseases of the Nervous System (1881), pp. 857–8, speaking of the treatment of neuralgia, recommends Duquesnel's aconitia in doses of .0005, gradually increased to .0013 if necessary, till relief be obtained, or till the characteristic peripheral numbness occurs.

Personal experience. Influenced by Prof. Gubler's article and by his books, I began using the aconitia of Duquesnel in the winter of 1877–8, with most gratifying results. More of the drug was imported, and in a few months several of my friends were trying the remedy—among them I may name Dr. McBride and Dr. Andrew H. Smith.

At a meeting of the Therapeutical Society of New York, held October 11th, 1878, I presented the report of the Committee on Neurotics of that Society upon the use of this aconitia. We reported ten cases cured or relieved. This report will be found in the *New York Medical Journal* for December, 1878. See also p. 367.

Since that time aconitia has been used by many physicians in numerous cases of trigeminal neuralgia, with very favorable results. A large proportion of cases have been cured, and some very ancient cases (8 to 12 years) greatly relieved by the medicine. A few cases only have been uninfluenced.

In the last two years the alkaloid has been offered in pillular form by several reliable drug-firms, and I can testify to the potency and reliability of Caswall & Hazard's tablets, and of Schieffelin's pills. These firms furnish doses of .0003 and of .0006.

In my first use of aconitia I employed a solution made by the late Dr. William Neergaard, the only pharmacist who then (1877–8) held a sample of Duquesnel's preparation. My formula was:

℞	Aconitiæ (Duquesnel's)	.006
	Glycerinæ,	
	Spt. vini rect.,	āā 4.
	Aquæ menth. pip.,	ad 62.

Each teaspoonful (estimating seven teaspoonfuls to 30.cc) contained about .0004. This dose was to be given two, three or more times a day, on an empty stomach, till the pain ceased or the physiological symptom—numbness—was produced. As my subject to-day is not clinical therapeutics as much as posology, I pass by many interesting facts about the use of aconitia, and omit all cases.

The remark which I have already made about the necessity of giving small doses of potent drugs to a patient whom we see for the first time, and of estimating his susceptibility, applies with especial force to aconitia. Bearing this in mind and carrying it into practice, we may be very bold, almost rash, later on, without running real danger.

Those of us who introduced aconitia in 1878 soon discovered that some persons, females especially, were powerfully affected by minute doses. Dr. A. H. Smith reported a case to our committee in which a lady was distressed by .00016, and I myself, while in a reduced state of health and suffering severe trigeminal pain, was severely benumbed by .0003 (though long afterward, when quite well, it required two doses of .0006 to produce nearly similar effects).

It is well, consequently, to give debilitated, susceptible and female patients, doses of .00024 or .0003 to begin with. These facts have induced the Messrs. Schieffelin & Co. to cease making pills of .0006, and to furnish only .0003, pills, which can be repeated at will. Messrs. Caswell & Hazard still furnish both doses in the shape of soluble tablets.

In a case of neuralgia, after a day's testing with minute doses, if I find no undue susceptibility to the drug, I give it freely—.0006 every three or four hours until distinct numbness and coldness (subjective coldness) be felt in the limbs and face. Then a longer interval may be allowed before giving another dose. Some subjects will take three or four tablets of .0006 each day, and be in a constant state of numbness without harm, and often with curative effect.

In some of my cases of chronic epileptiform neuralgia I have kept patients under the influence of the drug for days and weeks, —and have seen no evidence of cumulative effects.

As a rule, in testing a man of average physical development and not reduced by disease I at once start with doses of 0.0006.

As regards maximal doses, I may state that in certain cases of posterior spinal sclerosis with severe fulgurating pains I have given from 4 to 8 doses of .0006 each in 24 hours, producing in some cases faintness, sickness, and a considerable prostration. I might add that this form of nerve pain has never been relieved by aconitia, and that with hardly an exception, all the tabetic patients I have experimented on have not shown any trace of the numbness which is *the* sign of aconitia effect in healthy persons.

As a rule, the pain of trigeminal neuralgia ceases when the physiological effects of the drug are manifest. I do not pretend, and Prof. Gubler did not claim, that aconitia is a certain or specific remedy against trigeminal neuralgia, but it certainly is the best of all our present therapeutic resources against this terrible disease. Of course in certain cases, special etiological factors must be considered, and other treatment given besides the aconitia: for example, in clearly malarial neuralgia, and in syphilitic neuralgia, or in the (rare) neuralgia from bad teeth.

III.—PHOSPHORUS AND PHOSPHIDE OF ZINC.*

(*Phosphorus.*—U. S. P.)

Doses given by authorities on materia medica and therapeutics:

STILLÉ and MAISCH. National Dispensatory (1880), p. 1072. These authors, apparently wholly relying upon Gubler and Thompson, state that the dose varies from .003 to .005. They say: "Those who have most advocated its use recommend that a first dose of .003 should be repeated every four hours till six doses are taken. If then no improvement (in neuralgia) have occurred, the dose should be increased to 0.005, and repeated in the same manner as before."

They do not, however, mention Thompson's alcoholic solution of phosphorus.

Zinc phosphide (p. 1546) in doses of .004 to .008, and even .02.

STILLÉ. Therapeutics (1874), vol. i., p. 800. "Moderate doses of .0015 to .015." (Phosphorus).

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* The equivalent of zinc phosphide ($Zn_3 P_2$) is 195.6 + 62 — 247.6. Consequently one part of the phosphide contains 25 % (about) of phosphorus.

Wood, Therapeutics (1880) p. 113, recommends a mixture containing oleum phosphoratum, each dose to contain from .002 to .004; or of a chloroformic solution in a mixture, .004.

The dose of zinc phosphide he gives as .0006 to .0012 which is in strange contradiction to his full doses of phosphorus.

Bartholow. Materia Medica (1880), p. 96.

Dose of oleum phosphoratum, U. S. P., 5 to 10 drops (equal to .003 or .005).

Quotes Radcliffe's formula for pil. phosphori, .0018 in each pill. Also quotes Thompson's tinctura phosphori in doses equivalent to .0015 and .003.

The dose of phosphide of zinc is .005 to .015.

Rice. Posological Tables (1879). Oleum phosphoratum (p. 54). No dose given. Recommends Dr. Squibb's solution: Phosphorus, 1 part; cod-liver oil, 99 parts.

"Phosphorus, .0006 to .003, increased with caution."

Nothnagel and **Rossbach.** Arzneimittellehre (1878), p. 200. Dose from .001 to .005.

Gubler. Leçons de Thérapeutique (1877), pp. 236,7. Dose, .001 in granules; from 2 to 10 a day.

Praises the oleum phosphoratum in capsules.

Zinc phosphide, from .01 to .06 *per diem;* he rather depreciates its virtues.

Doses as given by authorities on clinical medicine:

Hammond. Diseases of the Nervous System (1881), p. 69. Speaking of cerebral congestion, he says that the oleum phosphoratum may be given in a mixture in doses of 5 drops.

Zinc phosphide, the formula of which he gives as Zn_3 P, and estimates as being one-seventh phosphorus, he recommends in .006 dose, in pill form (this gives .001 – of phosphorus); the phosphoretted resin may be used to make pills, each containing .001 + of phosphorus.

Flint. Practice of Medicine (1881), p. 797. Merely names phosphorus as a remedy for neuralgia; gives no doses or estimate of its value.

Anstie. On Neuralgia (1871), p. 180. States that he has used the phosphuretted oil and pills of phosphorus (Dr. Radcliffe's), containing .002, three times a day. He does not estimate it as especially useful. (This was written before the publication of Thompson's work.)

J. Ashburton Thompson. Free Phosphorus in Medicine, London (1874), p. 190: "The chief precaution to be observed in the treatment of neuralgia with free phosphorus * * * is to administer a full dose of the remedy in the first place."

" * * * unless .03 or more be given in the course of each twenty-four hours, frequent failures, or only partial successes in treatment will be met with." "But the remedy must be given in not less than this dose, *i.e.*, .005 repeated every four hours, from the beginning of treatment."

Page 191. He admits the utility of the alcoholic and ethereal solutions, reduced phosphorus, and even zinc phosphide, but he has had the best results from .005 of phosphorus dissolved in cod-liver oil, every four hours.

Thompson has more recently furnished the following formula for the preparation of a solution of phosphorus, which is not unpalatable to most patients.

Take of

Phosphorus, - - - - -	.06
Absolute alcohol, - - -	20.
Dissolve with heat	
Glycerine, - - - - -	48.
Alcohol, - - - - -	8.
Essence of peppermint, - -	1.2

Mix the two solutions, which make nearly 80.; one teaspoonful = .003. This should be given without water.

Personal experience. Very soon after the appearance of Dr. Thompson's article, I caused this solution of phosphorus to be made by Mr. F. Haas, by Caswell, Hazard, & Co., and by the late Dr. Neergaard, and used it a great deal. A weaker preparation or imitation, under the name of elixir of phosphorus, one teaspoonful of which contains .0015 is also sold, but I prefer the stronger form, and write for *solutio phosphori* (Thompson).

I have employed this solution with the greatest success in trigeminal neuralgia, and with some success in other neuralgias —following Thompson's plan of giving full doses, usually one teaspoonful (about .003, if we estimate a teaspoonful to be about 4 cc.), every 3 or 4 hours. I have known a severe facial neuralgia (not chronic epileptiform neuralgia) cured in two days, and even in 24 hours; several cases, in a week.

In conditions of nervous prostration, cerebral anæmia, incipient cortical degeneration (dementia), in melancholia, I have been much pleased with a combination of Thompson's solution and cod-liver oil in the proportions of 1 : 6 or 1 : 4, a teaspoonful of the mixture being given after each meal.

In other cases I have had an extemporaneous mixture made and given two or three times a day: Thompson's solution, 1 teaspoonful; sherry, 2 tablespoonfuls; cod-liver oil, from 1 to 2 tablespoonfuls; and the yolk of one egg, thoroughly beaten and mixed, with the addition of a little extra oil of peppermint. This is well received by most patients, and constitutes a most valuable tonic.

The phosphide of zinc in doses of .01 to .015 combined with nux vomica or with belladonna, according to indications, has seemed of some efficacy in the treatment of posterior spinal sclerosis, of cerebral anæmia, of nervous prostration ("neurasthenia"), and of incipient dementia.

With pills of pure phosphorus, I have had little experience. The pills offered by most of our manufacturing drug concerns are of too small a dosage. As may be seen from the citations made, and from my own experience with other preparations, the giving of .0006, or even of .0012, of phosphorus is of probably very little use. From .002–003 should be administered three times a day, with, of course, due watchfulness for signs of gastric irritation.

IV.—Crystallized Nitrate of Silver.

(*Argenti nitras.*—U. S. P.)

Doses as given by authorities on materia medica and therapeutics:

Stillé and Maisch. National Dispensatory (1880), p. 237.

Dose from .01 to .015 three times a day. Doses of .03 occasion no special symptoms, but larger quantities are apt to cause gastric heat, pain and nausea.

Stillé. Therapeutics (1874), i., p. 367, *et. seq.*

Quoting authors upon diseases of the nervous system he refers to doses varying from .01 to .015, three times a day.

Wood. Therapeutics (1880), pp. 51–3.

Dose from .015 to .03, in pill form, given upon an empty stomach, when it is desired to affect the stomach, and after meals, when the constitutional effects of the drug are desired.

"When given for a chronic disease, its administration should be suspended for one week, at the end of every third week, and its employment should not extend over a longer time than three months, without a protracted intermission."

BARTHOLOW. Therapeutics (1880), pp. 213–5.

Gives elaborate directions and formulas for its use in various visceral affections, dyspepsia, gastritis, colitis, etc., but hardly refers to its use in nervous diseases, and does not recommend it.

RICE. Posological Tables (1879).

Dose .015 to .12; increased with caution.

NOTHNAGEL and ROSSBACH. Arzneimittellehre (1878), p. 113.

In pill form, .005–.03.

GUBLER. Leçons de Thérapeutique (1877), p. 579.

Thinks that no good effects are to be expected from its internal use, and gives no doses.

Doses as given by authorities on clinical medicine:

The older English physicians, Sims, Wilson, Harrison, and Roget, quoted by STILLÉ, Therapeutics, i., p. 367, gave doses of .06–.36, three times a day, for epilepsy. It is not now employed for this disease I believe.

FLINT. Practice of Medicine (1881), p. 476.

Speaking of locomotor ataxia, he recommends giving from .01 to .02, three times a day, for several weeks; then suspending its use for a while.

HAMMOND. Diseases of the Nervous System (1881), p. 633.

In the treatment of locomotor ataxia, merely mentions dose of .015 three times a day; gives no details, and does not seem to attach any value to the drug.

WUNDERLICH, *Archiv der Heilkunde*, 1861, ii., p. 193 (cited by STILLÉ, p. 368), gave .012 twice and thrice a day; quoted by TOPINARD, he gave .01 three or four times a day; for locomotor ataxia.

BOUCHUT (cited by STILLÉ, pp. 368, 9), *Bull. de Thérap.*, lxiv., p. 57, gave to a child five years old, with paraplegia, .006 twice a day. To adults for paralysis from .024 to .06 a day.

TOPINARD, De l'ataxie locomotrice, Paris, 1864, pp. 435–468, gives a full account of the attempts to cure sclerosis of the posterior columns by silver; relates several cases of his own, and concludes that the drug is usually useless in locomotor ataxia; he gave from .01 to .09 *per diem*.

He gives the following doses as prescribed by several well-known physicians:

CHARCOT and VULPIAN in 1862 gave doses of .01, .02, .03 three times a day. Later CHARCOT has given as high as .08 in a day.

PIDOUX, .08 *per diem.*

GUBLER and BEAU, .10 *per diem.*

HILLAIRET, .15 *per diem.*

W. ERB. Ziemssen's Cyclopædia, Am. ed., vol. xiii. On Diseases of the Spinal Cord, pp. 614, 5. Recommends from .01 to .02 three times a day, or from .06 to .09 *per diem*, until 8. or 10. have been consumed. He has a high opinion of the medicine, for he says, p. 614: "Among the *internal remedies* for tabes, nitrate of silver undoubtedly stands first, as it can show quite undoubted results."

Personal experience. I have employed nitrate of silver extensively in the treatment of locomotor ataxia, and am almost disposed to agree with Erb. I can most positively assert that in quite a number of my cases the course of the disease has been materially checked, and in many others repeated periods of relief secured by nitrate of silver.

I have also given it in various forms of subacute and chronic myelitis, but with less definite results; though my impression of its action in these cases is favorable,

I seldom prescribe less than .0125 of silver at a dose, and usually give .03. The remedy is made up in a pill with an indifferent extract (taraxacum), or with extract of nux vomica, or with extract of belladonna, according to the indications of the case, whether for spinal stimulation or for sedation.

I always give the pill before meals, three times a day, and occasionally administer a fourth pill at bedtime. A course of silver, with me, usually lasts two months, which, at the rate of .10 a day, would give 6.—a perfectly safe quantity as regards danger of discoloration of the skin. After an interval of two or three months I often give another, shorter course, and so on.

None of my patients has as yet shown discoloration (argyria), and I have seldom been annoyed by the occurrence of gastric and intestinal irritation. Albuminuria from its use I have never seen.

CASE OF INJURY TO THE MOTOR AREA OF THE BRAIN, WITH EXHIBITION OF THE PATIENT.*

WM. M. G., æt. 27, Middletown Springs, Vt. Dr. Middleton Goldsmith. Nine years ago struck by a stone just above right ear. Was senseless. Patient says he could walk when he came to, but could not use left arm; claims that he could not move any portion of arm or hand. Could talk. Not much trouble from wound, but at the end became unconscious and had a convulsion.

Dr. Thomas was then called. Was convulsed generally, and had a depressed wound above the right ear; face perhaps a little paralyzed, and leg was a little weak, but the palsy of arm was complete. Several repeated spasms; was bled for convulsions, but no operation was performed; no bone ever came away; it was several months before wound healed. Had other convulsions in a week, then very frequently, daily, several a day, sometimes a week without attack; once was three weeks without attack. Seizures now quite frequent; also has petit-mal, and in this perhaps the hand is stiffish. No evidence of nocturnal attacks.

Marked analgesia of hand and fingers, though he says he feels contact of objects, and pin going through.

Hand very athetoid; was contractured in flexion for quite a while after wound, time uncertain; limber for 4–5 years.

The man's head exhibits a rounded, saucer-shaped depression in the middle of the right parietal bone. The lower edge of the cicatrix is eleven centimetres above the apex of the tragus, and its upper edge five centimetres from the median line. From the nasal spine to the anterior edge of the scar is sixteen centimetres. The diameter of the depressed area is between three and four centimetres, and its greatest apparent depth about eight millimetres. It is firm and bony throughout. Projecting the lines of Broca on the head the scar is found just anterior to the Rolandic line, overlying the middle of the ascending frontal gyrus.

The patient was examined by several members, who expressed the opinion that the man's epilepsy might be cured by the proposed operation of trephining.

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* Read before the American Neurological Association, June 21st, 1882. Reprinted from the *Journal of Nervous and Mental Disease*, 1882.

The patient was trephined by Dr. H. B. Sands, June 27th, 1882. From his report* the following notes are taken:

A nearly circular aperture 4.5-5 cm. in diameter was made. Depressed bone was found which was quite vascular and considerably thickened, being 12 mm. in its thickest part.

March 25th, 1883, the patient was reported in better condition, the seizures less violent, headache less intense, and the weakness of the right hand less marked.

REMARKS ON THE FREQUENCY OF HEADACHE AND CHOKED DISC WITH TUMOR OF THE BRAIN.†

WITH reference to headache I am ready to admit that it is one of the important symptoms, and in some cases that it is almost the only symptom; that it is excruciating and peculiar. It so happened, however, that in two of my cases of well-defined cortical sarcoma no headache was present. In the first case the symptoms began in the leg. There was no headache until one night the patient had an apoplectic attack. The lesion was situated in the paracentral lobule, and in the tumor there was a cyst. There was no headache that could be attributed to the sarcoma. In the second case of sarcoma there was no headache before the appearance of definite symptoms of brain tumor, but the patient experienced occipital neuralgia.

With reference to the neuritis and choked disc, I am obliged to confess that I have had a very singular experience. I have seen a number of cases of encephalic tumor and a number of cases of cerebral tumor. It has so happened that only one case of cerebral tumor, strictly speaking, had choked disc.

This was a case ‡ I had in 1880, which, during life, had a left hemiplegia and hemi-epilepsy, with chief symptoms in the arms and hand.

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* N. Y. *Medical Journal*, April 21st, 1883, p. 427.

† Part of the discussion by Dr. Seguin of a paper entitled "Notes on Twelve Cases of Brain Tumor, chiefly with Reference to Diagnosis," by Chas. K. Mills, M.D. Read before the American Neurological Association, June 22d, 1882. *Archives of Medicine*, August, 1882. The discussion appeared in the transactions of the association for that year. From the *Journal of Nervous and Mental Disease*, July, 1882.

‡ Case reported in full, p. 495.

An autopsy revealed a large sarcoma in the upper, middle part of the right ascending frontal gyrus. The presence of choked disc was verified by Dr. Amidon.

All of my cases of basilar tumor have had choked discs. Also, a case of sarcoma within the medulla oblongata. In a case* of abscess located in the frontal lobe there was no paralysis and no aphasia, the symptoms being those of extreme pressure. The pulse was slow—58, and lower at times. The optic nerves were somewhat congested, but there was no actual choking.

A case of sarcoma of the upper part of the left, ascending frontal gyrus, near the longitudinal fissure, which presented, during life, hemiplegia and hemi-epilepsy with chief symptoms in the foot, had, up to day of death, no choked discs.

A sarcoma of the left hemisphere, † developed in the paracentral lobe, pressing inward, which produced its first symptoms in the right foot—epileptiform attacks and paresis—later right hemi-epilepsy and hemiplegia, no aphasia, produced no choked disc. (Verified by Dr. Amidon). There was no headache in this case till a hemorrhage occurred from the exceedingly vascular growth.

In a case of angio-sarcoma of the right, ascending frontal gyrus (other tumors present) with first and chief symptoms in the left hand and arm, there was no choked disc. (Verified by Dr. Birdsall.)

In a case of sarcoma from the pia, penetrating the right sphenoidal lobe, the tumor being easily enucleated, there were marked pressure symptoms but no choked disc seventy-two hours before death.

In a case where a node pressed upon the upper part of the ascending frontal and parietal gyri, near the longitudinal fissure, there was left hemiplegia, first symptom clonic convulsion in left foot and leg, and typical choked disc.

I present these cases bearing upon the subject of choked disc not with the idea of lessening the importance of that symptom but with the intention of emphasizing the idea that we should not reject the diagnosis of cerebral tumor on account of not finding choked disc.

With the permission of the Association, I will make black-board illustrations of two of my cases of cerebral tumors. In one of the cases there was typical choked disc and excruciating pain; the lesions were in approximately similar regions, and nearly of the same size. Both tumors were globular. In both cases the first and principal symptoms were in the hand and arm. Yet in one case there was typical choked disc, in the other the optic nerves remained normal.

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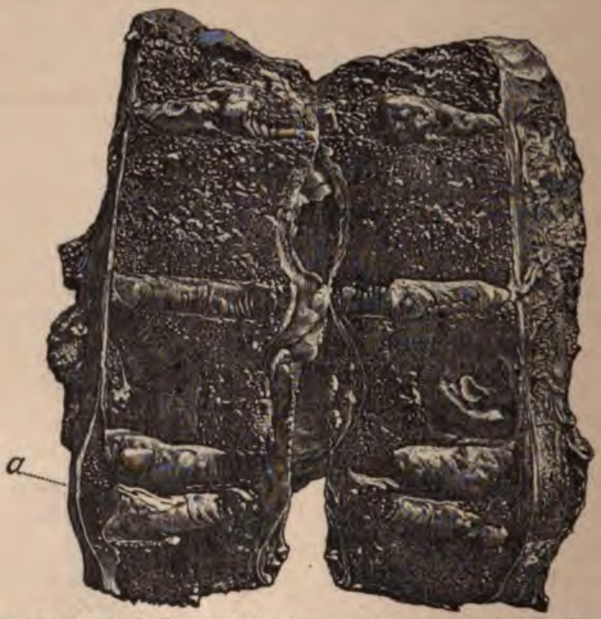
* See p. 452.

† See p. 499.

VERTEBRAL CANCER AND PARAPLEGIA.*

Mrs. P., aged over forty years, was seen with Dr. Burlingham, at Plainfield, New Jersey, on Nov. 10, 1881.

More than twenty years ago, while bathing, was struck in the left breast by a friend's elbow. Felt a good deal hurt at the time, and afterward said to this friend, "If I ever get cancer of the breast I'll blame you for it." No attention was paid to the small induration which remained. In the spring of 1879 this lump grew and caused some pain. Dr. Burlingham, and Dr. Willard Parker of New York, advised its removal, which was done by Dr. Hart in the same year. The tumor was examined



Longitudinal section of the bodies of the lumbar vertebræ, showing at *a* the elevation caused by the absorption of a vertebral body, the consequent approximation of cartilages and projection into the vertebral canal of the intervening tissue.

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* Read before the American Neurological Association, June 22d, 1882. Reprinted from the *Journal of Nervous and Mental Diseases*, July, 1882.

by some one for Dr. Parker, and was reported to be "myxosarcoma."

In a few months a marked recurrence of the disease took place in the breast, with involvement of the axillary glands. A second operation was done by Dr. Hart in June, 1880 and everything removed; it was a remarkably clean operation.

Since that operation there has been only a small varying (?) tumor near the anterior axillary fold. Patient was married in the autumn of 1880. Soon became pregnant and seemed well.

In December, 1880, driving home through the snow, was exposed to a cold wind, and both her feet wet. She fancied that the cold air "struck her left hip." In about two weeks began to complain of pains about the region of the left anterior superior spine of the ilium and below. Later had pain in right anterior femoral region.

In January, 1881, had a first attack of spasm in the back. The head was thrown back, and the lower part of the back was tense, painful, and causing the body to be thrown backward. She continued to have more or less of this "drawing" feeling in the lower part of the back, often as low as the sacrum. Then she could not stoop or bend forward.

Before her confinement early in February the pain appeared in both anterior femoral regions. This was a sudden attack of pain with "drawing" in the back; the pain then increased, with spasm or cramps, throughout the hips, thighs, and feet. These attacks often lasted half an hour. Forceps were used in the confinement; it was otherwise normal.

In the spring she seemed better; went about on foot, but had the same cramp pains developed early in the morning by turning in bed. No numbness. In July was at the sea-shore quite wretched. In August Dr. Burlingham went to see her in consultation with Dr. Risk. She then had more pain in the hips and back; lay in bed, afraid of any movement (of the left leg especially). Could not bring her heel to the ground.

Toward the end of September there was gradual loss of power in the right leg, then complete palsy of the left leg, and lastly the right leg was completely palsied; at the same time there were loss of feeling in the legs, and partial retention of urine.

Six weeks ago (about October 1st) the paraplegia was complete, with anæsthesia, but no abnormal reflexes, and has so remained. Has lost color and weight progressively; no fever (?).

Complete extension (or rather the attempt to do this) causes severe tearing pains deep in the abdomen, above the umbilicus. Has a pseudo-cincture feeling above the umbilicus. Of late there have been some reflex movements in the legs.

Examination.—The patient exhibits complete anæsthesia below a level 3 cm. above umbilicus in front, and as low as the posterior superior spine of the ilium behind. All voluntary power is lost below the epigastrium. Abdomen much distended; impacted fæces can be felt in the ascending and descending colon. Pricking causes reflex movements. There are no symptoms above the umbilicus, except the axillary tumor (quiescent nodule).

The spine presents two deformities: First, a well-marked kyphosis composed of three vertebræ in the lower dorsal region. Below this the spine is displaced anteriorly, and below; in the lumbar region there is another kyphosis.

The pains have ceased for several weeks. No alteration in nutrition; some œdema of the paralyzed limbs. Bladder quite full (now micturates by reflex action). In August, Dr. Risk found a little albumen and some casts; none since. Nurse reports thirst and flushing of cheeks in afternoon. Reflexes all raised.

Diagnosis.—Pott's disease in lower mid-dorsal region, probably from cancer of the vertebræ and of the dura mater; compression of the spinal cord.

Clinically the symptoms are those of Cruveilhier's "*paraplégie douleureuse.*" Advise no treatment. If pains return morphia to be given freely.

On December 30th I recived the following letter from Dr. Burlingham:

"Dear Doctor: The patient, Mrs. P., you saw with me died yesterday morning. There had been no very material change in her condition since you saw her, except a gradual and general loss of strength. The appetite was good, and digestion well performed. The distension of the abdomen was much less, and the 'drawing' pains had almost ceased. Urine passed sometimes without her knowledge, and about two weeks ago was very bloody for a couple of days. The evening temperature ranged from 37.8° C. to 40° C.; morning temp. nearly normal. For the past month there has been a very considerable reflex action in the legs, the left one more violently; and she complained of her feet aching.

"Bed-sores formed over the trochanters and the sacrum. Forty-eight hours before death she first complained of stiffness about the jaws. Clonic spasms now followed, involving the facial and throat muscles, and causing a fear of choking to death. These continued till about half an hour before death.

"A *post-mortem* examination was made about eight hours after death.

"The spinal column only was examined. I send herewith the portion removed for your inspection.

"We did not find the bodies of the vertebræ in the condition in which you regarded them at the time.

"The mental condition of the patient remained clear until within fifteen minutes of death."

Had not Dr. Burlingham taken the trouble to send me the vertebræ the case might have remained an anomalous and discouraging one for the student of spinal affections. The bones viewed externally after death seemed nearly normal.

The portion of the vertebral column sent consisted of two segments, one from lower dorsal and one from the lumbar region.

In both these portions, several bodies contained round masses of grayish gelatinous cancer, some nearly 2.75 cm. in diameter, quite destroying the cancellous body. At two points, one in the dorsal region, there was absence of an entire body, with projection of the anterior wall of the vertebral canal upon the spinal cord, causing compression of that organ; this was at the 10th dorsal. The adjacent bodies had come together, causing the kyphosis observed during life. A similarly total destruction of a vertebral body had taken place in the third lumbar vertebra, causing some pressure upon the cauda equina. The smaller nodules were of varying size and age; all, however, gelatinous and tending to the classic globular form. There was no trace of cancer in or about the cord.

A microscopic examination of the spinal cord showed no distinct secondary degeneration, but a well-marked, diffused myelitis. This finding, with the fact that the projection of the remains of the 10th dorsal vertebra in the canal was small, makes it probable that the spinal cord suffered more from an irritative process than from simple compression.

MYELITIS FOLLOWING ACUTE ARSENICAL POISONING (BY PARIS OR SCHWEINFURTH GREEN).[*]

THE physician who, meeting with a case of arsenical paralysis, would seek for information on the subject in the accessible and contemporary treatises upon diseases of the nervous system, would be grievously disappointed. Such writers as Grasset, Ross, Wilks, Bauduy, Hamilton, do not mention the affection at all; the illustrious Romberg and Erb merely give it a passing reference. Prof. Hammond (1881) in the last edition of his treatise, says nothing of paralysis following acute arsenical poisoning, and refers to paralysis and anæsthesia as results of slow poisoning. Apparently he has seen no cases of arsenical paralysis. Rosenthal (1875) devotes only a short paragraph to arsenical nervous symptoms; refers to paralysis in the course of chronic poisoning. In a case which he saw there were paralysis, partial anæsthesia, and diminished electro-muscular contractility. Leyden (1875) in his classical work on diseases of the spinal cord,[†] gives a *résumé* chiefly after Leroy D'Étiolles.[‡] He does not appear to have had cases of his own, and considers the disease a neuritis.

A little more extended research in older books, and in periodicals, brings to light numerous observations and some valuable experimental studies upon the subject.

Indeed, arsenical paralysis seems to have been very early noticed, and to have attracted considerable attention until within the last twenty years. As early as the thirteenth century P. Abano[§] refers to paralysis and contractures after arsenical poisoning. These symptoms are also mentioned by Forestus[‖]

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[*] Read at the meeting of the Medical Section of the New York Academy of Medicine, October 17th, 1882. (The original cases alone had already been read before the American Neurological Association, at its Eighth Annual Meeting, June 21st, 1882.) Reprinted from the *Journal of Mental and Nervous Disease*, vol. ix., No. 4, October, 1882.

[†] Klinik der Rückenmarks-Krankheiten, Bd. ii., p. 296.

[‡] *Gazette Hebdomadaire*, Tome 4, 1857, pp. 141–144.

[§] De venenis eorumque remediis, cited by Imbert-Gourbeyre, *l. c.*

[‖] Cited by Imbert-Gourbeyre, *l. c.*

(about 1560–70); and Zacchias[*] (1630) mentions paralysis, spasms, contractures, and anæsthesia as following poisoning. From that time arsenical paralysis is frequently mentioned by medical writers. Hahnemann,[†] in one of his earlier works, (1786), relates several cases.

In 1812, Sir Benjamin Brodie, in an interesting communication to the London Royal Society, entitled "Observations and experiments on the actions of poisons on the animal system," devotes a section to the effects of arsenic, and relates how in several of his animals (rabbits and dogs) the hinder extremities became paralyzed. He considered the brain to be affected in these cases.

The following interesting case was published, in 1809, by Dr. G. Thilenius.[‡]

A young lady having observed a hard lump in her left breast, neglected it until the ensuing spring, when it became very painful. A miserably ignorant barber, who was consulted, applied a preparation of arsenic. This was followed by ulceration and increased pain, and, according to the father's statement, three days later her arms and legs became insensible, and so much paralyzed that she could neither walk nor feed herself. The limbs were also cold. In the course of two months the arms recovered, and the legs improved steadily. Electricity was used; the tumor removed by the knife. At various times there occurred prickling and jerking in the legs. Anæsthesia and atrophy not mentioned.

In about a year after the attack the patient was able to walk without a cane; her limbs were warm, and the wound in the breast well healed.

In 1793 (three years after the attack) patient was perfectly well, married, and had a child.—Obs. No. 80 of Leroy D'Étiolles.)

Orfila,[§] the great French chemist and toxicologist, in experiments upon dogs, made prior to 1840, noticed paralysis of the hinder extremities in dogs which survived arsenical poisoning (also in fatal experiments).

Prof. Christison,[‖] of Edinburgh, in his classical work on poisons, treats of symptoms of arsenical poisoning in a masterly way. He makes three categories of cases of arsenical poisoning. In a first class of cases, in which, with symptoms of violent inflammation of the gastro-intestinal tract, death results in from twenty-four hours to three days; nervous symptoms not present. In a second class of cases, with little evidence of inflammation, extreme prostration and syncope are the chief symptoms, death occurring within six hours; no paralysis observed. Convulsions may close the scene. In the third category, that of subacute cases, there is moderate gastro-intestinal inflammation; symptoms are same as in other classes, but milder. In the later stage these cases are apt to show marked nervous symptoms: coma, epileptoid attacks, mania, tetanus, hysterical seizures, partial paralysis resembling lead paralysis in affecting the extremities; contractures may exist. In speaking of symptoms connected with irritation of the *primæ viæ*, Christison makes this shrewd remark, which applies critically to many of the older cases of arsenical paralysis: "Cramps in the legs and arms (occur in arsenical poisoning), a possible concomitant of every kind of diarrhœa."

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[*] Quaestiones medico-legales, Romæ, 1621–50, cited by Imbert-Gourbeyre.

[†] Ueber das Arsenik-Vergiftung, Leipzig, 1786, cited by Imbert-Gourbeyre.

[‡] Medic.-chirurgische Bemerkungen, Frankfurt, 1809, in Leroy D'Étiolles, p. 63.

[§] Traité de Toxicologie, Paris, 1852.

[‖] A Treatise on Poisons, Phila., 1845, p. 244, *et seq.*

The father of modern clinical medicine, Graves * (1842), after speaking of paraplegia from inflammation of the bowels, refers to Orfila's experiments in which all (?) the dogs which survived arsenical poisoning were paralyzed in their hinder limbs, and states that in his opinion in cases of arsenical as well as of lead poisoning, the poison acts directly on the central nervous system (spinal cord), and that the palsy is not due to the intestinal irritation.

Huss,† of Stockholm, in his work on alcoholism (1852), mentions several cases of arsenical poisoning with severe nervous symptoms. He gives one which is instructive as regards its etiology.

For the cure of intermittent fever, a large teaspoonful of Fowler's solution was given at one dose (equivalent to .035 of arsenious acid). After the usual symptoms of acute intoxication, there gradually ensued an almost complete paralysis of the extremities, with anæsthesia of the hands and feet, severe pains and cramps in the lumbar region and lower extremities.

In 1857 we meet with quite an important contribution to this subject. Leroy D'Étiolles, ‡ in his work on paralysis of the lower limbs, devotes a chapter to arsenical paralysis, and relates the following three cases (in addition to the case of Thilenius already quoted).

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* Clinical Lectures, Gerhard's edition, Phila., 1842, p. 94.

† Cited by Imbert-Gourbeyre.

‡ Des paralysies des membres inférieurs, deuxième partie, p. 28, *et seq.*, Paris, 1857.

Obs. 79.—Poisoning from external application of arsenious acid ; general paralysis ; recovery of upper extremities first.

Male patient . . . aged. Dr. Trochon, of the hospital at Pornic, amputated his leg for cancer. In the cicatrix cancerous buds appeared. Arsenical paste, made one hundred times too strong by druggist's error, was applied, and very soon symptoms of acute intoxication appeared; life saved with difficulty. At the end of ten days patient convalescent, but with well-marked paraplegia and paresis of the arms.

Seen five months later by Leroy: Arms weak and not adroit; tendency to drop wrist. Marked paralysis in remaining lower extremity, with emaciation, but not positive atrophy of muscles; contracture in semi-flexion; foot hyperextended (pes equinus); toes flexed. Sensibility to touch and pain much impaired on limbs. Electrical tests not used.

Gradual improvement of paralysis in spite of progressive cancerous infection.

This case bears a certain resemblance to my own cases. Although it is stated that the muscles were not atrophied as in lead paralysis, yet from the contractures and the degree of emaciation present, it seems to me highly probable that there was atrophy, widely distributed, as in mild cases of poliomyelitis. It is a pity that electricity was not used, although at the time when this observation was recorded, 1855, only the bare fact of diminution or loss of faradic contractility could have been determined.

The contracture in flexion with pes equinus is strikingly like what existed in my own Case 3.

Obs. 81.—Poisoning by the ingestion of arsenious acid; paresis of arms; paraplegia lasting fourteen months.

A female patient aged thirty-seven years, was admitted to the service of Dr. Bouvier, Hospital Beaujon, January 22, 1850, suffering from severe toxic symptoms produced by eating cakes charged with arsenous acid.

As soon as the urgent symptoms had subsided (time not noted), it was discovered that the patient was paralyzed in her lower limbs, and that they were the seat of painful jerking (reflex movements ?). Her arms were weak. On 18th February, on leaving the hospital, she was unable to stand, and said that she could not feel the floor under her feet. In September of the same year she was readmitted with pleurisy; and it was noted that while her arms had recovered, her legs were just as weak, and as insensible to touch. At no time was there interference with the functions of the rectum or bladder. Later some improvement took place, but the patient finally died of exhaustion caused by a profuse diarrhœa. No autopsy.

Obs. 82.—Case of Aran in *Union Médicale*, July 6, 1852. On June 9, M.

Aran presented to the Société Médicale des Hôpitaux, one of two young men who had, two months previously, been poisoned by arseniate of sodium. The victims had swallowed this salt, supposing it to be tartrate of sodium. One died in twenty-four hours; and a lady to whom they had given some of the poison, is not yet perfectly well.

In the surviving male patient interesting nervous symptoms have appeared. In about fifteen days after the ingestion of the drug, symptoms of paralysis appeared in the lower limbs, more marked in the right leg. The upper extremities have also been weak. The paralysis has remained very much in *statu quo*. The paralyzed parts are somewhat anæsthetic. The lower limbs are the seat of tingling below the knees; and the upper extremities in the finger-tips. At one time the paretic extremities showed diminished calorification. General health good.

M. Duchenne examined the young man and found slight diminution of electrical irritability, and the skin showed diminished sensibility to the current.

Later, on 8th September, M. Aran reported to the Society that the patient had recovered, apparently in consequence of forty-six baths and forty-six douches at Bagnères de Luchon (hot sulphur springs). Improvement showed itself distinctly after the thirty-sixth bath.

Leroy makes these general statements: In lead paralysis the forearms are usually affected (sometimes only one); arsenical paralysis tends to involve all the limbs; the lower limbs are more affected; often there is well marked paraplegia; the action of the bladder remains normal. Sensibility is usually much impaired (nearly as much as motility). He refers to wasting of muscles, but states that it contrasts with the positive atrophy of lead paralysis. Electro-muscular contractility persists, but is diminished. Treatment is efficacious, and the duration of the paralysis is usually less than one year.

Shortly after the appearance of Leroy D'Étiolles' work, a learned French physician, Imbert-Gourbeyre,* professor at the medical school of Clermont-Ferrand, published a series of articles in the *Gazette Médicale* (1858), in which he gave an elaborate account of our previous knowledge of arsenical paralysis. I am indebted to this essay for bibliographical data. The articles contain nothing original. In 1863, Smoler † published a case of paralysis after acute arsenical paralysis, which is referred to by Rosenthal (1875).

Jaccoud ‡ (1864) devotes several paragraphs to arsenical paralysis (paraplegia), and expresses his belief that the palsy is caused by the direct action of the metal or its compounds upon the tissue of the spinal cord. He does not, however, appear to have seen a case.

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* Études sur la paralysie arsénicale. *Gazette Médicale*, 1858, pp. 5, 19, 59, 94.

† Lähmung nach Arsenikvergiftung. *Œsterreich. Zeitschr. für pract. Heilkunde*, 1863.

‡ Les paraplégies et l'ataxie du mouvement, Paris, 1864, p. 323, *et seq.*

In 1881, Seeligmüller[*] placed on record four cases; two after acute poisoning, and two other chronic intoxication. In his acute cases he noted paralysis, numbness, and anæsthesia (in toes), contractures, wasting of the extensors especially. The paretic and wasted muscles showed fibrillary contractions; the nails were gradually lost, Electro-muscular contractility was diminished or even lost.

He gives the following points for differential diagnosis from lead palsy: the acute origin of the paralysis, disorder of sensation as well as of motion, rapid muscular wasting, absence of blue line on the gums, and of cachexia.

In the same year appeared the essay of Popow,[†] of St. Petersburg, upon the pathological anatomy of arsenical paralysis as produced artificially in animals. Popow carried on his experiments under the guidance of Prof. Mierzejewski; giving arsenious acid to dogs in doses ranging from .003 to .12 at a dose, producing acute and chronic intoxication.

In cases where death ensued in four or five hours after ingestion of the poison, the spinal cord showed both macroscopic and microscopic lesions. The gray matter appeared swollen, intensely red, more especially in its two enlargements. Microscopic examination revealed enlargement and congestion of the small blood-vessels, and accumulations of lymph corpuscles in the lymph-spaces.

There was also abundant extravasation of blood-corpuscles and plasma around the vessels, especially in the central portions of the gray matter. The walls of the blood-vessels were in a state of fatty degeneration.

The ganglion cells exhibited three degrees of change. A first degree of alteration showed cells well stained by carmine, and containing vacuoles of variable sizes, some of which could be traced into the cell-processes. A second form of cells had no processes, were feebly colored by carmine and exhibited a punctate granular infiltration. Lastly, here and there were cells in a third state of change, consisting only of a nucleus surrounded by dark brownish pigment. The white substance only showed pigment masses here and there, more especially about the blood-vessels.

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[*] Ueber Arseniklähmung. *Deutsche med. Wochenschrift*, 1881, No. 14, *et seq.*

[†] Ueber die Veränderungen im Rückenmarke nach Vergiftung mit Arsen und Blei. *St. Petersburger méd. Wochenschrift*, 1881, No. 36.

In cases of acute intoxication in which a fatal result ensued in the course of three, five, or six days, the distinction between the white and gray substances of the spinal cord was less defined. The vascular injection and the exudation of plasma were less marked, but on the other hand the changes in the ganglion cells were more distinct; the vacuoles larger, and the granular state more pronounced. There were more cells, or properly remains of cells, of the third category above described. The white substance was normal, except some enlargement of blood-vessels, and considerable accumulations of pigment.

In the chronic cases, those in which death occurred in the course of three months (one animal had paresis of the hind legs not long before death), the spinal cord appeared less firm, and the microscopic appearances differed noticeably from those observed in the acute cases.

The walls of the blood-vessels were much thickened, and showed a distinct fibrillary structure, with diminution of the calibre of the vessels, and exudation of blood in the perivascular spaces. In the meshes of the perivascular spaces were extensive hyaloid masses. The number of ganglion cells was much diminished; those remaining showed large vacuoles, and belonged to the first group described. In these cases the white substance was much more affected, especially in the postero-lateral columns. The cylinder-axes exhibited points of swelling here and there; they were granular; in many preparations they were merely represented by groups of fine granulations. The septa of the white substance likewise exhibited a granular change; and the periphery of the white and gray substances was thickly strewn with small masses of black pigment.

The spinal nerves, carefully examined at their origin, and at various points of their course and distribution, presented no pathological alterations.

From these *post mortem* observations Dr. Popow concludes that:

1. Arsenic, even in a few hours after its ingestion, may cause distinct lesions of the spinal cord, of the type known as acute central myelitis, or acute poliomyelitis.

2. In the more chronic cases the pathological changes are found in the white as well as in the gray substance, constituting a diffused myelitis.
3. The peripheral nerves remain normal, even three months after intoxication.
4. The paralysis of arsenical poisoning is of central origin.

It might be added that in three guinea-pigs poisoned by lead, and dying on the sixth, seventh, and tenth days, similar lesions were found, *i. e.*, evidences of more or less diffused myelitis, and no lesions of peripheral nerves.

This essay, issued under the supervision of so distinguished a neurologist and microscopist as Prof. Mierzejewski, is in many respects the most important contribution to the subject. Connecting its conclusions with inductions which can be legitimately drawn from the cases of Leroy, Seeligmüller, Smoler, and Rosenthal, and my own, we are able, I think, to form a definite conception of the true nature and relations of arsenical paralysis.

In last year's *Philadelphia Medical Times* Prof. J. M. Da Costa * relates a case of subacute myelitis which occurred in a man who had been taking "small pinches" of arsenic (arsenious acid?) for three months. The general features of this case and the paralytic phenomena are so unlike what has been observed in the other cases referred to in this paper, that I entertain a doubt as to its having been an "arsenical paralysis." The rapid improvement under very large doses of iodide of potassium, and the history of a venereal sore one year before admission, would seem to furnish a better clue to the nature of the myelitis.

The cases which have fallen under my own observation are three in number. The subjects were all would-be suicides with Paris green,† and they presented remarkably similar symptoms. In many respects the cases resembled those already related.

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* Clinical Lecture on Arsenical Paralysis, *Phila. Med. Times*, Vol. II.; p. 385, 614.

† Paris or Schweinfurth green is a compound substance which is best designated as aceto-arsenite of copper.

In looking up this point I was astonished to find that such a popular and so constantly used a term as Paris green, was not to be found in the indexes of any of our dispensatories, treatises on materia medica, and, stranger still, not in works on toxicology.

Prof. Chas. F. Chandler, in reply to a note, very kindly gave me all necessary chemical information on the subject.

CASE I.—Samuel L., hostler, seen March 21, 1879, in consultation with Dr. M. Burke.

At the end of January, while in good health, swallowed a large quantity of pulverized Paris green. Had much difficulty in swallowing it, and very soon was led to a drug store, where emetics were given ; and later he was taken to Bellevue Hospital, where the stomach-pump was thoroughly used. Vomiting, gastric pain and irritation, extreme prostration, lasted four or five days.

Soon after he began to go about his room, he noticed numbness in his fingers and hands, followed in two or three days by similar sensations in his feet. Paresis appeared about the same time in all the extremities, and had steadily progressed to extreme paralysis below the knees, with wasting of the muscles there. Has had much burning, gnawing pain in soles and insteps ; a little in the hands. Ten days ago became unable to stand. No cerebral symptoms, or palsy of bladder, or jerking of legs.

Examination.—Hands and forearms only weak ; no positive paralysis or atrophy ; no anæsthesia.

Legs completely paralyzed below the knees ; cannot move feet or toes. Thigh muscles are weak. Marked atrophy of calves and of anterior tibial muscles. No anæsthesia of soles, unless it be a slight tactile dullness.

Test with faradic current ; no reaction in right leg, nerves, or muscles. In left leg no reaction in anterior tibial muscles or nerves, but a feeble contraction can be produced in the calf.

Patient is at times hysterical.

At fifteen had a chancre, not followed by secondary symptoms.

I have no further notes of the case ; but some sort of galvanic treatment was carried out. A few months afterward I learned that the patient was well, and some time in the winter of 1880–81 he came to my office and exhibited a vigorous pair of legs. He had completely recovered and was at work again as a hostler at Jerome Park.

CASE II.—Mary N., aged sixteen years, was admitted to the New York Hospital on December 11, 1878, in the service of Dr. Woolsey Johnson. To Dr. R. W. Amidon, then house physician of the hospital, I am indebted for notes of the case and for the opportunity of studying the case in its later stages. Dr. Johnson has kindly given me permission to use the case.

The patient was a strong, rosy-cheeked girl of German parentage. She had never suffered from rheumatism or malaria. Thirteen days before admission she swallowed five cents' worth of Paris green. In five minutes she vomited, and after an emetic had been given she vomited again, rejecting all (?) that had been swallowed. Probably had some gastro-enteritis, as she vomited and purged for two or three days.

It is reported (by patient and her friends) that on the first night she had fever. Second day, no fever or pain. Third day, at three P.M., had fever for one hour and a half ; burning pain in toes ; hands felt stiff. On the fifth day, at nine A.M., fever returned, with slight headache, but no chill. The burning pain extended up to the knees. One week after taking the poison her legs became stiff, and she lost power in her arms ; had "cramps" in her hands. These symptoms continued, but the headache ceased, tried to walk, but found that she was partially paralyzed in her legs ; needed help to walk, and suffered pain in her knees (in the attempt). Three days later (tenth day) loss of power increased ; had cramps in hands ; had tightening sensations in hands and feet, and they began to peel and showed a mottled red and white appearance. Bowels and bladder normal.

Has not been unwell for twelve weeks ; previously regular.

Condition on admission, thirteen days after taking poison : Patient complains only of headache, and of inability to walk, because "cords of knees are stiff." Appetite, bowels, bladder, and eyes normal.

Hands are cold and moist. The extensor muscles of both hands are weak, those of right hand weaker. Some twitching of long flexors and of interossei.

The skin is lax. The small muscles of the region of the right little finger are completely paralyzed and wasted. The right thenar eminence is smaller than it should be. The hypothenar group on the left side is in the same condition. Grasp very weak. On dynamometer each hand shows about 20° (on outer circle). There is hyperidrosis. No anæsthesia is present, but she complains of a burning when pricked with a pin.

The legs are semi-flexed, showing mostly palsy of the extensors of both feet. The left foot is more inverted than the right. Legs and thighs smaller and colder than normal. Toes are red ; the circulation is sluggish. The muscles of the legs are not flabby, but the anterior tibial regions are flattened. Hamstrings rigid on both sides.

Circumference of right thigh, 30.5 ; right leg, 23.5.
" " left " 30.5 ; left " 24.5.

There is complete paralysis of the anterior tibial muscles. The peroneal and posterior tibial groups are somewhat atrophied and paretic. Great toes are motionless. Flexion of thighs is moderately good ; extension complete. No increased reflex actions. Is rather hyperæsthetic (in legs).

Dec. 14th. A tendency to retention of urine is noted (but is not again referred to). Examination with the faradic current showed good contractions in left thenar and hypothenar eminences, but none in the right. In the right leg there is slight reaction in the anterior tibial muscles ; none in the peronei.

Dec. 26th. It is noted that there is no faradic reaction in the anterior tibials and peronei. Sensibility is good. Hamstrings less rigid. Patient has plaster apparatus for legs, and the application of the faradic current.

Jan. 11, 1879. Walks with some support, and has done so for a week. Left leg nearly straight.

Jan. 15th. Circumference of right thigh, 34.0 ; right leg, 24.
left " 34.5 ; left " 26.

Toes always cold and moist ; tender to slight pressure. Less contraction of hamstrings. No reflex actions in legs. Interossei of hands do not improve, and remain as flaccid, atrophied, and weak as on admission (faradism not used on upper extremities).

Jan. 24th. Galvanism tried for first time; ten cells cause contraction of tibialis and peronei ; eight cells (Stohrer battery) cause contraction of quadriceps, sartorius, and muscles of calf. Some atrophy of extensors of the right forearm and hand ; good reactions (current not stated) in ulnar distribution, but not in wasted extensors. On the left side good reaction in forearm and hand, except abductor pollicis, to both galvanism and faradism.

Jan. 27th. Menses appeared with great pain.

Feb. 4th. Electrical applications omitted because of *malaise*. Patient doing nicely; muscles react with small amounts of electricity.

Feb. 9th. Some trophic changes in feet; nail of big toe coming off. The skin is rough, and there is vaso-motor disturbance. Reactions improving (in legs).

March 10th. Walks quite well. Some remaining weakness of anterior tibial muscles.

Discharged improved.

This patient at once began to be treated as an out-patient at the Manhattan Hospital, and after several weeks of treatment by galvanism mostly was completely cured.

The notes taken at this time have been misplaced, but our recollection is clear that her upper extremities were about well, though perspiring, and that the lower limbs exhibited paresis, a sluggish circulation, and a peculiar sensitiveness and tenderness. She was able to walk alone, but lame. Her general health was quite good.

It seems certain that this was a case of subacute poliomyelitis chiefly. The inflammatory action must have extended to deeper parts of the gray matter, as shown by continued hyperalgesia and by the contractures.

Case III.—Ellen R., aged twenty-six years, admitted to Manhattan Hospital May 10, 1881. In September, 1880, took a large dose of Paris green. Was exceedingly ill; vomiting and diarrhœa. In a week nearly complete paralysis developed. Legs completely paralyzed; forearms the same.

A gradual recovery began in the course of a few weeks (no treatment).

Three weeks before admission to the hospital, Dr. J. B. Emerson, who visited her in the country, found her fingers and the soles of her feet nearly insensible to pricking. I examined the patient May 9, 1881, and the following notes were taken. Can walk with a little aid, impeded by moderate contracture of right knee, and tenderness of feet. No voluntary power (motion) below ankles. Complete anæsthesia to contact on soles of feet and on finger-tips. Feels cold and heat, however, and pricking quite well. The upper extremities simply present a slight paresis with moderate wasting of the hand muscles, some interossei quite wasted, and some large fibrillary movements in the same. No cutaneous trophic changes. Thighs moderately wasted, with some contracture of right hamstrings. No patellar tendon reflex. Calves and anterior tibial muscles are much wasted; legs and feet bluish and cold; slight tactile anæsthesia of feet.

Patient is thin and in poor health: has been using an unknown quantity of morphia.

She was ordered a mixture containing diminishing amounts of morphia; and Dr. Adam, assistant physician of the hospital, applied galvanism and faradism very faithfully to her for weeks. He also gave her passive movements and massage. The improvement was steady, and in a few weeks patient left the hospital almost perfectly cured as regards paralysis, and in good general health. She was forty-eight days in the hospital. The day after admission Dr. Adam made a thorough testing of the affected muscles with the galvanic and faradic currents, which may be summed up by saying that most of the paralyzed parts exhibited the degeneration reactions, viz.: 1, diminished or wholly lost faradic reaction in muscles and nerves; 2, sluggish contractions to galvanism, with ancc=cacc in many muscles, and ancc > cacc in some. For example, in the muscles of the leg below knees a very strong faradic current caused no reaction. In the right gastrocnemius ancc > cacc. In other muscles cacc=ancc. In some interossei of hands ancc>cacc.

This patient is again under my care at the Manhattan Eye and Ear Hospital (October, 1882), for the cure of the only remaining weakness, viz.: paralysis of both anterior tibial muscles causing pes valgus. This is the only muscle which does not respond to the will, but all the muscles of the leg show a most astonishing quantitative reduction in electrical reactions; no reaction in muscles or nerves to full strength of faradic secondary current, and few small reactions in nerves and muscles to fifty good Leclanché elements. Reactions obtained are of normal quality.

In this case, besides the contracture of the hamstrings, as in Case II., we have distinct though slight anæsthesia to indicate a certain extension backward of the lesion, in the spinal gray matter.

To sum up, these three cases presented evidences of slight subacute, diffused myelitis, more distributed in the anterior cornua. In Case I. the symptoms were more purely those of poliomyelitis.

In all cases the symptoms of myelitis followed within a week after the ingestion of the poison.

If we compare the symptoms present in the various human cases related and quoted, and the pathological appearances found by Popow in his animals, it is, it seems to me, legitimate to reach the following conclusions:

1. Arsenical paralysis is the expression of a myelitis.
2. This myelitis approximates the type known as poliomyelitis in so far as the symptoms are chiefly motor; that the paralyzed muscles undergo some atrophy, and exhibit the degeneration reactions to electrical currents; that the bladder is never palsied; and that in animals the ganglion cells of the anterior horn are extensively diseased.
3. There is usually more than poliomyelitis, as shown by Popow's *post-mortem* findings, and by the presence in living

human subjects of pains in the nerves and muscles of the affected limbs, and by the occurrence of actual anæsthesia.

4. Consequently it might be better to speak of arsenical paralysis as due to diffused central myelitis with special involvement of the anterior gray matter.

5. Whether this myelitis is strictly arsenical, *i.e.*, caused by the direct effect of the arsenic on the tissue of the spinal cord, or whether it is produced (as are many forms of myelitis) by the irritation of peripheral nerves (cutaneous, intestinal and gastric nerve-endings), is a question which cannot at present be definitely solved, but which presents an interesting field for future research and speculation.

ON A PECULIAR CUTANEOUS LESION (ULCUS ELEVATUM) OCCURRING DURING THE USE OF BROMIDE OF POTASSIUM.[*]

IN the last few years, more especially since the publication of Trousseau's lecture entitled "*Exanthèmes sudoraux,*" [†] much attention has been paid to toxic or medicamentous eruptions.

Among these, the cutaneous lesions produced by the various bromides, when taken internally, have been particularly well studied, and quite a variety of eruptions have been observed and recorded by dermatologists and neurologists.

In the last edition of Prof. Duhring's excellent work [‡] we find a section devoted to this subject, and the following bromic eruptions are described under the head of *dermatitis medicamentosa.*

1. Acne-form pustules.
2. Brownish discoloration of the skin.
3. Simple papular eruption.
4. Confluent or molluscoid acne.
5. Maculo-papules.
6. Carbuncular acne.
7. Bullæ.
8. Rupia.

Ulcers are not named in this list, and I do not know that they have ever been described, unless it be in the shape of isolated, ulcerated, carbuncular acne.[*]

It has been my fortune to observe this year two cases of large, elevated ulcers upon the legs, occurring in epileptic patients using the bromide treatment, and I desire to place on record this new form of cutaneous lesion, probably medicamentous in origin.

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[*] From the *Archives of Medicine*, Oct., 1882.

[†] *Clinique médicale de l'Hotel-Dieu*, 2me êd., 1865, i, p. 199.

[‡] "Practical Treatise on Diseases of the Skin," 3d ed., Phila., 1882, p. 348.

[*] Dr. A. Voisin, in his monograph, "De l'Emploi de Bromure de Potassium dans les maladies Nerveuses," Paris, 1875, mentions a very similar lesion, which undoubtedly Dr. Seguin had overlooked.—[R. W. A.]

CASE I.—Miss C., aged 25 years, epileptic since her fourteenth year. The disease has been fairly well controlled by moderate doses of various bromides, under the supervision, in the last few years, of her physician, Dr. P. C. Barker, of Morristown, N. J. Very little facial acne has appeared, and at no time has severe bromism been apparent. Some ten years ago, while the patient was in Germany with her family, there appeared some "boil-like" sores upon the lower part of each leg, which soon coalesced into ulcers, one on each leg, presenting very much the same appearance as those of later development about to be described. On the left leg there was an "ulcer," also below the head of the fibula. In a few months these ulcers healed. So far as the mother of Miss C. knows, there was no increase in the amount of bromides, or change of bromide, prior to the appearance of the eruption, nor does she remember any special treatment, other than the application of some herbs to the sores, which determined their healing. The patient, continuing the bromide (various formulæ) treatment steadily, had no further serious eruption or ulceration, indeed no special bromic symptoms, until about fifteen months ago, when large, purplish papules, like "boils," appeared upon the outer side of each leg, about four inches above the external malleolus. These discharged, leaving ulcerated spots which coalesced, forming an ulcer which increased steadily in size until the early spring of this year, when I saw the patient. This account of the early appearances and development of the sores is derived from the patient's mother, whose qualifications as an observer are fair. Still, the lack of professional study of the early stages of the lesion is a *hiatus* much to be regretted. It is doubtful if the ulcer was ever a common excavated one; Mrs. C. thinks that for months prior to my examination its surface was distinctly raised above the level of the healthy skin.

When I saw Miss C., in March of this year, there was a large ulcer on each leg, almost encircling it, leaving a bridge of healthy skin over the anterior surface of the tibia. The rest of the leg, at a level about four inches above the malleoli, was covered by an irregular ulcerated patch, raised from 2 to 4 mm. above the skin. Its area was irregular, somewhat pear-shaped, with its largest development posteriorly. Its greatest width, vertically measured, was perhaps ·8 cent. The surface of this "ulcer" was different from any thing I had ever seen. As stated above it was raised quite uniformly above the healthy skin; its edges were abrupt, almost vertical, and showed no signs of cicatricial action. The elevated floor of the ulcer was firm, grayish-red in color, with here and there an adherent crust; it secreted a fetid, sanious, puriform liquid, and bled upon being touched with moderate violence. It did not look like ordinary granulation tissue; it was much firmer, composed of larger masses, and, in fact, at several points it presented a slightly villous or rather papillomatous appearance. There was no burrowing of pus under the ulcer, and the rest of the leg was normal—perfectly free from "boils" or papules. This description, insufficient as I feel it to be, applies to both legs; the ulcers upon them were almost precisely alike; the lesion was a symmetrical one. (On the left leg, just below the head of the fibula, was an irregularly-shaped, smooth, white and coppery cicatrix of the ulcer which appeared ten years ago, and healed in a few months.) The patient's face and neck presented hardly a trace of bromic acne, and I was told that her body was free from ulcers or other eruptions; she showed none of the other symptoms of bromism, and her epileptic attacks were still recurring in a mild form occasionally.

I was so much impressed with the firmness and elevation of these patches that I could not avoid a suspicion of epithelioma, in spite of the symmetry of the lesion which indicated its toxic or systemic origin. Prof. Henry B. Sands, to whom I then sent Miss C., decided that there was nothing malignant about the ulcers, and he looked upon them as eruptions connected with the patient's condition or with her medicines, and he sent her to Prof. William H. Draper, who also looked upon it as medicamentous eruption. Dr. Draper has recently written me as follows concerning the case :

". . . I remember Miss C.'s case perfectly, even though I have no notes of it. . . . I examined some scrapings from the ulcer and found only granulation cells. I think it was unquestionably a bromic acne. The lesion begins, I believe in the follicular structures, it is said in the sweat follicles, but I suspect in the sebaceous ones as well. . . ."

Acting upon Dr. Draper's suggestions, Dr. Barker applied pyroligneous acid to the ulcers and they at once began healing. I saw the patient a fortnight ago and found the ulcers almost entirely healed, with copperish smooth cicatrices such as we see after common ulcers of the legs.

It is interesting to note that the bromide treatment was continued unchanged.

CASE II.—Annie L., aged twelve years, was brought to me at the Manhattan Eye and Ear Hospital, on February 24, 1882, to be treated for peculiar attacks which I considered epileptic, and of a form intermediate between petit-mal and psychical epilepsy. In many seizures there were hallucinations of vision. The first attacks, which appeared about eighteen months ago, after a fever, were distinctly spasmodic. In the last few months more attacks of both sorts; right internal strabismus has appeared. Much complaint of headache, principally over the right eye; grasp of right hand less strong than that of left. Further details concerning the neurosis are unnecessary for my present purpose.

The child's health seemed good; she was fairly well-colored, and her skin was everywhere normal.

She was put upon our regular bromide treatment, taking from three to four grammes a day at various times. The indications of coarse cerebral disease were so strong that I also gave her .75 gramme of iodide of potassium three times a day, and applied a few blisters behind the ears.

The epileptic attacks were at once controlled, and the child seemed to bear the medicines well. Owing to some annoyance at having to wait very long one day, the mother ceased bringing the child to the hospital; this was some time in March, and there was then no bromism or eruption.

Rather accidentally, Annie again came under my care in June, by being brought to my clinic at the College of Physicians and Surgeons. I at once recognized her, and upon inquiry found that she had had private treatment since leaving the hospital, and had taken bromide of potassium only (no iodide). The attacks had been few and slight, more of the psychic order: fear of falling down stairs, seeing animals, weeping, calling out to mother, and complete amnesia of attack. She was somewhat stupid from the bromide, but the amount given was impossible to ascertain. There was almost no facial acne; the child's color was clear and healthy. The mother, however, stated that since leaving the hospital clinic, the child's legs had become the seat of very painful sores, which she wished me particularly to examine. Bandages were removed from both the child's legs, and my surprise may be imagined when I beheld ulcers precisely like those of Miss C., seen a few months before.

The lower part of each leg, some 5 cent. above the malleoli, was encircled by a large ulcerated patch distinctly elevated above the surrounding healthy skin. The outline of the sores was very irregular, varying in vertical width from 3 to 8 cent.; the largest surface of each sore being on the fibular side of the leg. The edges were sharply defined and nearly vertical. The surface of the sores, raised 2 or 3 mm. above the healthy skin, was covered with brownish-black scabs and most offensive sanies. On removing some of these scabs a rough granulating surface, easily bleeding, was revealed. I use the word granulating, but the appearance was that of a firmer, more villous, in places almost papillomatous formation, than the delicate translucent and uniform surface of ordinary granulations.

The mother, a not over-intelligent Irish woman, stated that this local trouble began in April, while using the bromide prescribed by the physician she consulted after giving up the hospital. At first the right leg was affected with large pimples or boils, which "broke," ulcerated, and coalesced into an open sore. In about a fortnight the left leg was similarly affected. It is interesting to note that this woman's account of the beginning of the ulcers agrees precisely with that given by the very intelligent mother of Miss C.

I directed that the ulcers be gradually cleaned of scabs by frequent washing, and that a strongly carbolized ointment be used twice a day. The dose of bromide of potassium was fixed at two grammes night and morning. The mother, having other children to attend to in the midst of the difficulties of tenement-house life, did very little toward cleansing the sores, and did not apply the ointment as carefully as necessary. Besides, the child dreaded to have the legs dressed, and cried violently each time they were washed. I was away a great part of the time in the months of July and August, and during that time there was no material change in the size or appearance of the ulcers. The child remained free from ordinary bromism, and her complexion was good. The seizures occurred but rarely.

On July 28th I gave her ether, and after removing nearly all the scabs from the left ulcer, I applied to it Paquelin's cautery quite freely, and ordered a carbolized lotion for a few days, to be followed by applications of balsam of Peru ointment. At the same time I cut out a strip of the ulcer and adjacent sound skin for histological study; this was pinned on a cork and placed in bichromate of potassium solution. In cutting away this little flap I demonstrated that there was no appreciable lesion of the subcutaneous connective tissue; the derma seemed hypertrophied, but the whole piece was easily dissected away with the scalpel. I reduced the bromide one gramme night and morning, and gave five drops of Fowler's solution after each meal.

I saw the child again Aug. 21st. The ulcer which I had cauterized—that on the left leg—was nearly half healed over; the right leg presented the same appearance as at first, and in spite of my urgent orders, had not been well cleaned of scabs and sanies. I directed that to be done before the child was brought again, intending to use the cautery again, or to apply nitric acid. The same amount of bromide was to be taken night and morning, and eight drops of Fowler's solution was to be given after each meal.

I have met with a third case, in which the legs were affected in a way not unlike what the mothers of Cases 1 and 2 describe as the first or carbuncular stage of the ulcer; and probably, if the bromide treatment had been persevered with, ulcers might have formed in this case also.

Miss A. B., aged 17 years, a handsome girl, with a very fresh complexion, of German parentage, consulted me on November 1, 1880, for epileptiform attacks. She was a healthy child. At 8 years had an attack of typhoid fever lasting several weeks. On getting out of bed at the end of eleven weeks, found that she could not walk. It was nearly spring (fever in August) before she walked freely. Arms were unaffected; mind normal. In her ninth year had a convulsion one morning, biting her tongue. During the day legs became quite weak, especially the right. There seemed well-marked paralysis, but it is not certain whether there was any muscular wasting. Under galvanism and strychnia she gradually regained the use of her legs, but ever since she has had convulsions at very irregular intervals—every two or three weeks, or at intervals of months. In the last two years three or four severe attacks; last one in September. Has had occasional doses of bromide, but no systematic treatment. Patient states that in night attacks she wakes dizzy, has time to call some one, hears a loud noise as of a wheel going faster and faster, until she loses consciousness; hears no voices or bell-sounds. A cousin, who has witnessed seizures, says that there is a severe convulsion, in which patient's eyes are open; after attack, she is stupid, and wants to rise; talks and weeps violently. Then she falls into a heavy sleep.

Last summer, was exposed to severe solar heat and had a profuse nose-bleed. Ever since has been liable to petit-mal; a whirling dizziness, followed by "faintings" in some instances. Further questioning shows that ever since first spasm she has had a third sort of attack, consisting in the sudden appearance of "balls before the eyes," followed by temporary diplopia; no drowsiness.

In 1877, Dr. Brown-Séquard was consulted, and gave Miss B. his bromide mixture. This was faithfully employed for six months. The patient then had large sores with scabs upon her legs, mostly on right. It is difficult to ascertain whether these sores were like those described above, or discrete scabbed sores, such as I have seen in other patients, and termed rupia-like. There was no facial acne, and the epileptiform attacks were suspended. The bromides were stopped and the sores quickly healed.

Examination showed some anæmia and symptoms of gastric catarrh. The case was so clearly epileptic, that in spite of the former bad effects of bromides, I persuaded the patient to try them again; giving her my solution of chloral and bromide,* three teaspoonfuls (twelve grammes), at bedtime, well diluted. Treatment and diet were also ordered for the gastric catarrh. In eight days a "boil" appeared on the right leg, constituting a small abscess, which ruptured spontaneously on the twelfth day, near a scar of the former eruption three years ago. Dose of bromide solution reduced to two teaspoonfuls at night. On the fifteenth day several large indurated pimples had appeared around the small abscess. On the nineteenth there was quite a crop of large purplish papules with evident tendency to suppuration on the right anterior region. This appearance, the patient said, was identical with that observed while using the Brown-Séquard mixture three years ago, and she begged me to stop the bromide. I might add that for a week or ten days arsenic and sulphide of calcium had been given to control the eruption. There was no bromism, hardly a trace of common facial acne, and the gastric symptoms were better.

I suspended the use of ordinary bromides, and gave bromide of zinc, bromide of camphor, digitalis, valerian, etc., at different times; also, at times, renewed tonics and treatment for the dyspepsia. The epileptiform attacks have been almost perfectly under control, and there has been no return of the eruption (which passed away in a week or ten days after the bromides were withheld).

Very probably, had I persevered in giving the solution of bromides, the papules would have all undergone suppuration and ulceration, coalesced, and formed an ulcer more or less like those observed in the other cases.

The third case, though incomplete, has this value, that it bears out what the mothers of Cases 1 and 2 claimed as to the early appearances of the ulcers, viz.: at first an acne, undergoing suppuration and ulceration; the resulting ulcers merging into one sore.

The following points in the clinical history of these ulcers are interesting:

1. Their origin in acne.
2. Their progressive and semi-malignant tendency.
3. The absence of bromism in the patient.
4. The absence of slight development of common facial acne at the same time.
5. The possibility of curing them by energetic local treatment (Case 1) without omitting the bromides.
6. The inefficiency (?) of arsenic and of calcium sulphide (Case 3).

Histology.—Sections of the piece of tissue removed from the ulcer show great increase in the thickness of the rete

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* Chloral hydrate, 15; potassium bromide, 30; water, 200 grammes.

Malpighii, with hypertrophy of the whole skin in places. In several places villosities visible to the naked eye occur, made up of all the elements of the skin thrown up and out into a minute mushroom-like or polypoid mass. In other places proliferation of young cells has taken place in the cutis, with atrophy and rupture of the epidermal layer, and partial escape of the newly-formed tissue, constituting a sort of abscess, opening externally. In other localities the patches of inflammation were wholly circumscribed, and sub-epithelial. The deeper layers of the skin, and to a certain extent the subjacent connective tissue, are infiltrated at certain points with young cells. The papillæ, hair-follicles, and sweat-glands do not appear to be the seat of any primary or important inflammatory change. In no part of any section was its surface (edge) covered by granulation tissue, as in a common ulcer.

From these appearances we may conclude that the ulcer resulted from a dermatitis, which was partly suppurative, but largely hypertrophic.

A CASE ILLUSTRATING THE COINCIDENCE OF DISEASES: CERVICO-BRACHIAL NEURALGIA AND ANEURISM OF THE INNOMINATE ARTERY.*

THE influence of diagnosis, upon therapeutics and upon prognosis has seldom been more strikingly shown in my experience than by the following case:

Mr. S. S., aged fifty-two years, consulted me June 5, 1882, for a severe neuralgic ailment of the right side of the head, neck, and arm. He related the following history: Early in the summer of 1881, he had been thrown out of a carriage upon the sidewalk, but received no evident injury. After this fall he was restless and nervous, felt badly, had more or less gastric disorder. Spent the month of August in Saratoga, but was unrelieved. About that time he first noticed pain near the right olecranon process. This pain was quite localized at first, but soon later it extended toward the shoulder; very gradually increased in extent and severity, occurring in more frequent paroxysms. Late in the autumn the shoulder region was involved; and in December pain was felt in the head, a little to the right of the vertex, and later behind the right ear. Downward the pain has extended to the hand; the fingers have never been painful or numb. The pain has been somewhat nocturnal, but never periodic. The patient has suffered extreme agony for months, pain extending from the right parietal region down the neck to the right shoulder and arm. No treatment until December, then for two months Mr. S. was under the care of a specialist for diseases of the nervous system. Has recently been at the Hot Springs of Arkansas, where, with some internal treatment and applications of hot water in bags, he was somewhat relieved. Two weeks ago was subjected to strong electrical applications which greatly aggravated his neuralgia, particularly increasing the occipito-parietal pain. Since the electrical applications, the right arm has felt big and tight (not exactly numb). After paroxysm of pain the veins of the right arm appear full. A paroxysm occurs in my office, with chief acute pain behind right ear; it is evident that the patient suffers extremely; perspires during the attack. Exertion, use of arms, or walking, causes increase of pain, or even produces a paroxysm. Fortunately no morphine habit has become established. No syphilis.

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* From the *Archives of Medicine*, October, 1882.

Examination: Patient presents the usual facies of prolonged suffering; is pale and thin. Seat of pain as above stated; occipito-parietal, cervical, and brachial. Right pupil a trifle larger than the left. Nerve trunks not tender, but painful regions are hyperæsthetic in paroxysms of pain. There is no paralysis, anæsthesia, or muscular atrophy; the movements of the arm are free, except at the shoulder joint where some resistance and crepitation from false anchylosis.

The right radial pulse is very feeble, much smaller than that on the left side. The right carotid pulse is likewise much smaller than the left. The right hand is slightly swollen and tumid.

There is no sternal or pectoral deformity, but the supra-clavicular regions are both full, without yielding any unnatural pulsation. The heart is rather large, and at its base is a rough double murmur, which can be traced upward to a point of maximum intensity over the junction of the right second rib and sternum. No fullness or pulsation in supra-sternal notch. There is a slight hoarseness. Many of these points were determined at a second examination.

Taking into consideration the place of beginning of the pain, its distribution, and its typically neuralgic nature on the one hand, and on the other hand the absence of pain near the seat of intrathoracic disease, I made the double diagnosis of cervico-brachial neuralgia, and aneurism of the innominate artery. Contrary to what Mr. S.'s former medical advisers had said, I did not believe that his neuralgia was a sympathetic or reflex pain dependent upon the aneurism, and hence, incurable. I thought the coincidence a fortuitous one.

Acting upon this belief, I at once began treatment by cauterizing the neck with Paquelin's instrument, giving a deep injection of morphia over the brachial plexus, and ordering 4. of Thompson's solution of phosphorus (equal to about .003 of phosphorus) to be taken every three hours. In forty-eight hours very great improvement had occurred; no pain in the head since cauterization, and only two paroxysms in the shoulder and arm. Several cauterizations were made; morphia given by the mouth for a few nights; the arm was kept quiet. In about two weeks iodide of potassium was substituted for the phosphorus, and quinine also given. The neuralgia had almost ceased by the end of June; but the whole arm felt queer, heavy, and swollen (semi-painful); the radial artery was smaller, the substernal dullness more marked, and the double murmur over the innominate artery louder. In other words, while the cervico-brachial neuralgia was nearly cured, the aneurism was making progress.

Occasionally, there was slight return of cervico-brachial pain.

On August 23d, Prof. Austin Flint corroborated the diagnosis of aneurism of the innominate artery, and suggested a trial of Tufnell's rest and low-diet treatment. This the patient has decided to submit to. He has no neuralgia (none to present date, September 8th); he is troubled by a severe cough, with bronchial catarrh; his right arm is puffy and bluish, and feels badly. The local physical signs in the chest are the same. He takes quinine after breakfast, and a gramme of iodide of potassium in infusion of digitalis four times a day.

I am led to publish this case because of the belief that had I looked upon the neuralgia as sympathetic and expended my therapeutic efforts upon the aneurism as the *fons et origo mali*, I should have also failed to relieve the patient.

ANEURISM OF THE CŒLIAC AXIS.*

THE specimen I present to-night was removed from the body of a sailor, 36 years of age, who died on the third of May, in the New York Hospital. On admission, April 30, he gave the following history:

Two months previously he began to suffer from rheumatic pains in the legs, arms, and left side of body, which pains had been decidedly nocturnal. The pain in the abdomen he located in the epigastrium, going through to the lumbar region, around the left hypochondriac space; and he described it as very sharp and severe. He stated that until one month ago he never had any dyspnœa; that he never had suffered from acute rheumatism or received any injury. Five years previously he contracted syphilis, and since has had secondary skin and throat symptoms. He had continued the duties of an able seaman during the first month of illness, but had after that time taken to his bed. He noticed about three weeks ago that lying on his back caused increased pain.

On being examined in the ward, he was found lying upon his right side, his knees drawn up, his face being pale and expressive of pain and anxiety. The tibiæ and ulnæ were found swollen and tender; and the glands about the elbows and neck enlarged.

Great tenderness was found in the abdomen over the left hypochondriac region, the epigastrium, and over the lower ribs near the spine. In the epigastrium a pulsation was distinctly visible, and on applying the hand a little firmly, a distinct aneurismal thrill was perceived. Ausculation showed the heart and arch of aorta free from abnormal sounds, but about 3. cm. above the ensiform cartilage in the median line, a loud, hard systolic murmur was heard. On tracing it downwards, it appeared loudest at the apex of the sternal appendix, getting fainter below that point until lost midway between the umbilicus and pubes. In the back no true murmur was to be heard, but a sort of shock or "thud," with the systole of the heart, was audible over the tenth dorsal vertebra. No aneurism was to be found in any of the superficial arteries. A careful examination of the urine failed to reveal albumen or casts.

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* A specimen presented to the New York Pathological Society, May 8, 1867. Reprinted from the New York *Medical Record*, July 15, 1867.

This article is inserted here out of chronological order, being overlooked at the proper time, because it narrates a rare case and is on a kindred topic with the foregoing article.—[R. W. A.]

The diagnosis was made of aneurism of the aorta in the neighborhood of the cœliac axis. Preparations were made to put the patient upon the postural plan of treatment, and meanwhile morphia was given hypodermically to allay the pain. Unfortunately the issue was nearer than anticipated. On May 3, at 1 P.M., the patient put his hand upon the tumor and fell into a fatal syncope. No imprudent movement had been made previous to this.

The autopsy was made twenty-four hours post mortem. The abdominal cavity contained an enormous quantity of blood; 2 kilos. of clot, and 2,000 cc. of liquid blood. The heart and lungs were healthy.

The aorta was removed as far as its division in the pelvis. The valves were healthy, but the vessel was extensively atheromatous; in the arch anteriorly an aneurismal pouch had commenced to form. Lower down, opposite the cœliac axis, no dilatation was to be found, the branches of the vessel arising in a normal manner. But on tracing out these branches, a large aneurism was found connected with the cœliac axis proper, adherent on the left to the suprarenal capsule, on the right to the lobus Spigelii of the liver, and above intimately connected with the diaphragm. The branches of the axis escaped from the lower part of the sac, whose walls, 1 cent. thick, were rough internally, made up of very old layers of fibrin; the rupture having taken place (an opening 1 cent. across), at the anterior edge of the adhesion with the liver. The sac contained but a little liquid blood and clot. The remaining abdominal vessels were healthy.

The spleen, liver, and kidneys were healthy. The physical signs in the case are remarkable, and might have been sufficient for the making of an exact diagnosis. There was no doubt as to the height of the aneurism, and dilatation of the aorta might have been excluded on account of the absence of the of bruit in the back. It is very remarkable that the patient had never suffered from vomiting or any disorder of the digestive organs excepting constipation.

HYSTERICAL CONVULSIONS AND HEMI-ANÆSTHESIA IN AN ADULT MALE: CURED BY METALLO-THERAPY (GOLD).*

EXAMPLES of hysterical convulsions in the male sex are rather frequent in youth and boyhood, but after twenty they become so rare as to be worthy of record. Still more unusual is it (in this country at least) for hemi-anæsthesia to follow the succession of convulsions. For these reasons, and because the case presents points of interest as regards diagnosis and therapeutics, I desire to place it before the readers of the *Archives*.

James A., 21 years old, single, and a laborer by occupation, was brought to the Manhattan Eye and Ear Hospital by Dr. Smith, of Newtown, Ct., for the diagnosis and treatment of an alarming set of nervous symptoms, briefly summed up as convulsions, extreme staggering, left-sided hemi-anæsthesia.

History.—A year ago the patient fell from the upper platform of a freight car, a distance of at least ten feet, striking the ground upon the back of his head. He thinks that he was unconscious for a few minutes, but did not vomit. Remained well after this fall until some six weeks ago. Denies sexual excesses or irregularities. At that time, some six weeks ago, he had an ill-defined illness—apparently a severe "cold," characterized chiefly by pains all over his body, in the muscles mostly. Thinks that he had no fever (locality is malarious, however), and is positive that he had no articular swelling or sore throat. The account of the order of appearance of the nervous symptoms is obscure, as Dr. Smith did not see patient until two weeks ago. Then had already had several "fits," apparently of an epileptic nature; he was not paralyzed, but exhibited complete insensibility to pricking on the left side of his head, face, tongue and body. He also staggered somewhat. He complained of headache, near the vertex and over the right parietal region. Convulsions occurred every night; and one night about ten days ago there were several, which were witnessed by Dr. Smith. In these attacks the patient was stiff; eyes closed, showing, when the lids were raised, normal pupils; the respiration was slow and gasping; the spasm was only tonic, and lasted, quite certainly, not less than three minutes. There was no frothing of the mouth, or subsequent drowsiness. The patient claimed not to know anything of these seizures; attacks occurred yesterday. A friend of the patient describes attacks lasting an hour and a half. The staggering gradually increased during the fortnight of observation; clear (colorless?) urine was often voided; no globus or emotional seizures; has seemed rather obtuse or stupid. Much bromide of potassium has been administered. At first he had 2.75 every four hours, and later every two hours, and less often. Altogether has taken about 45 grammes in ten days.

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* From the *Archives of Medicine*, Oct., 1882.

Examination.—Patient is an average, dull-looking Irishman, generally pale, and with the neurotic white circle about his mouth strongly marked. Comes into the room supported by two persons; staggers preposterously; when not supported plunges off to one side or the other; no paralysis; sees and hears well (to simple tests); pupils normal; left side of body, face and tongue presents complete analgesia. Ends of fingers are a little sensitive to *deep* pricking (only in last two or three days). The various modes of sensibility and the special senses were not critically studied, because we purposed doing this on another day. To watch-test and to ordinary objects there was no deafness or blindness of the left ear and eye. An interesting experiment was made upon the patient as regards his equilibrium. I placed him in the middle of the room, loosened his friend's hold of his arm, and told him to look up at the ceiling and try to see certain fine marks upon it. Thinking that I was testing his eyesight, he strongly directed his attention that way and stood *perfectly well*, without a trace of his staggering; which, however, returned the moment that the test was over and he was told to stand alone—*that* he could not do (while thinking of it).

Without saying anything to the patient or to the physician and students standing by, I applied two twenty-dollar gold pieces to the patient's left hand, and afterward to his forearm, cheek and tongue. I most positively said or did nothing which could *suggest* anything to him. He could not tell whether I meant this as a continuation of the examination, or as a remedial measure; he looked and spoke as if he thought I was amusing myself in applying the gold.

In a few moments, one to three minutes, sensibility returned in each part where the metal had been laid; completely so in the tongue, and partially in the cheek, forearm and hand. The patient was amazed. I ordered a capsule containing .30 of citrate of iron and quinine, and .01 of extract of nux vomica, to be taken four times a day.

The next day, September 21st, the patient was examined by my colleague, Dr. W. R. Birdsall, who found him nearly free from staggering; pricking with a needle was felt a little less than normally on the left face, quite normally on the left arm and hand. On the left leg (not yet treated) pricking was somewhat felt, but simple touch was not perceived. A belt of gold plates was applied round about the calf for ten minutes, when sensibility was found to be restored, not simply where the plates had been laid, but throughout the extremity. On neither day was any phenomenon of " transfer " observed.

On Sept. 23d patient, claiming to be perfectly well, left the hospital, contrary to my request.

The diagnosis of the case presented but slight difficulties, in spite of its extreme rarity. The staggering was evidently overdone, or at least greater than in any organic or functional cerebral disease known to me; and it was made to cease by diverting the patient's attention in an interesting manner. The convulsions were too long to be anything but hysterical, and the state of the pupils indicated the non-epileptic nature of the seizures. The continuation of the symptoms—nay, their aggravation—under severe bromide treatment was in accordance with my own experience in hysterical cases.

The brilliant success of metallo-therapy in this case is interesting and very puzzling. I believe that every physician present when the gold was first applied, will agree with my statement that there was no sort of *suggestion* made to the patient—nothing was said until after the patient himself looked up in amazement at the restoration of sensibility to his hand and tongue.

It might be added that in the last two years I have had several successful cases of metallo-therapy in my practice—all of them reactions to gold. One of the most striking was that of a girl aged about sixteen, showing decided chloro-anæmia, but free from hysterical symptons except analgesia of the whole left upper extremity, and the neurotic white circle about the mouth. In this case an elongated oval area on the extensor surface of the forearm remained analgesic in spite of several short applications of the gold, but yielded to their continued contact for twenty-four hours.

TWO CASES OF GLYCOSURIA; ONE TRUE AND ONE SIMULATED.*

THE two following cases are presented because they are both in some way unique.

CASE I.—Mrs. —— consulted me in October of this year for simple, moderate dementia. She was fifty-one years old, and had masturbated for years. Six years ago she had an attack of quite acute melancholia.

In the last three years marked emaciation had taken place. No positive delusions or hallucinations seemed to exist. The memory had been fairly retained.

The patient fancied that her shoulders were paralyzed, and wanted to have her arms cut off. There existed no actual paralysis. The patient's manner was demented and hypochondriacal. She had no symptoms of diabetes except emaciation, hence it was not suspected.

As a matter of routine her urine was examined with the following result: An afternoon specimen had the specific gravity of 1,045, and contained a trifle over five per cent. of sugar, but was otherwise normal. A specimen passed the next morning had the specific gravity of 1,018, and did not contain a trace of sugar.

Oct. 14th.—Two days later, a morning and night specimen were sp. gr. 1,020 and 1,011 respectively, and contained no sugar.

Oct. 16th.—The urine had the sp. gr. 1,019 and 1,023, and was free from sugar.

Oct. 21st.—Four specimens were found of normal specific gravity and reaction. When last heard from (about November 12th) the patient was doing well and had no symptom of diabetes. The patient had neither medication nor diet which could have caused the sudden disappearance of sugar from the urine.

Cases of mild diabetes are not rare where an occasional sample of urine is found devoid of sugar; but a fall in the specific gravity from 1,045 to 1,018 and from five per cent. of sugar to none in twenty-four hours, is by no means common.

How long the patient had been passing sugar cannot be told, and we may perhaps look on the case as one where some peripheral sensory impression or some transient central alteration produced a disturbance of the chylo-poietic circulation which interfered in proper glycogenesis. This is rendered more probable because Pavy, Cyon, Aladoff, Schiff, and Eckhard have shown that irritation or destruction of many parts of the peripheral nervous system will, in a reflex way, produce glycosuria, while by puncturing the floor of the fourth ventricle, Bernard long ago caused sugar to appear in the urine of animals. Among diabetics, too, a profound mental or bodily shock almost invariably causes an increased excretion of sugar.

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* Reprinted from the *Archives of Medicine*, Vol. viii., No. 3, Dec., 1882.

CASE II.—In May of this year an old patient of mine sent three specimens of urine, with the request that they should be thoroughly examined. He said they were passed by a niece of his, who was under the care of a homœopathic physician for some hysterical affection. More definite data than these it was difficult to obtain. A night specimen of May 18th was pale, cloudy, contained some uric acid, and a *trace* of sugar. It had the sp. gr., however, of 1,070! A specimen passed the next morning contained only a trace of sugar, and had the sp. gr. of 1,048. A noon specimen of that day was pale, poured like syrup, and, with only about three per cent. of sugar, had the sp. gr. 1,090!

Besides these examinations, which were made by Dr. Amidon, I sent the heavy specimen to Mr. Charles Rice, of Bellevue Hospital, and the following is an extract from his letters in reply:

"There is, however, something present which I cannot make out exactly, but which I have separated and find to resemble in some of its properties 'peptone,' or digested albumen. If a portion of the urine is mixed with about five times its volume of absolute alcohol and shaken, a gummy substance separates, which may be caused to agglutinate into a lump by judicious turning of the test-tube. After being washed with alcohol it is found to be but little soluble in boiling water, but it dissolves in cold water, particularly after addition of a drop or two of hydrochloric acid, to a very thick, almost colorless, faintly opalescent liquid. It gives no reactions with nitrate of silver, or mercuric chloride, and does not exhibit any reactions of the ordinary gums."

June 6th.—Two more specimens were procured and examined by Dr. Amidon and Mr. Phelps. A night specimen was clear, pale, and gave a faint sugar reaction. It was syrupy, and had a sp. gr. of 1,105! A morning specimen, while it seemed to contain less sugar, had a sp. gr. of 1,117!! A fraud was immediately suspected, and its detection was attempted.

Mr. Rice kindly examined the latter specimen and said: "The last specimen of 'urine' seems to contain but little genuine urine, to judge from the small amount of the sulphates, phosphates, chlorides, and urea present." "It contains a body (or mixture of bodies) insoluble in alcohol, and partaking of *some* of the properties of starches and of gelatin, without, however, giving a reaction for starch itself, or a definite reaction for gelatin." "I should say the thing is a fraud."

A series of experiments were then made with gelatin, cane sugar, and various syrups. It was finally found that a mixture of a fine sorghum syrup and urine, in such proportions as to bring the specific gravity up to about 1,115, produced an exact imitation of our puzzling specimen. It gave the same precipitate to absolute alcohol, and contained the same amount of grape-sugar. It was, moreover, found by Dr. Amidon, that on standing and fermenting the cane sugar was transformed into glucose and gave a magnificent reaction to Fehling's test, while, when fresh, only a faint sugar reaction was obtainable. Also it was noticed that the specific gravity of the specimen of June 6th, which had stood in the laboratory uncorked for ten days, had fallen from 1,117 to 1,010, showing that the cane sugar had fermented and disappeared.

The mother of the patient was asked to *see* some urine passed in a clean vessel and send it for examination.

It was some time before this could be brought about, but at last a night specimen of urine was sent in which was clear, of a reddish-yellow color, acid, free from albumen and sugar, and of the specific gravity of 1,024.

NOTE ON CRANIO-CEREBRAL TOPOGRAPHY. ILLUSTRATED.*

THE surgical anatomy of the head, with reference to its contents has been developed with remarkable completeness within the last ten years, chiefly by the researches of Broca, Bischoff, Heftler, Turner, and Féré, by which cranio-cerebral topography has been firmly established as a branch of practical anatomy. Acting upon the data thus obtained, Broca, Lucas-Championère, Weir and others † have successfully trephined for the relief of aphasic and paralytic symptoms. The location of many convolutions and fissures of the cerebrum can be accurately mapped out upon the surface of the skull, or even upon a living head, by the projection of certain lines and measurements from certain points thus obtained, as well as from some natural landmarks.

For the projection of these lines, the head is placed in a particular position, as can be easily done when we operate upon a bare skull; but which can also be approximated when we deal with a living subject, either sitting up or lying in bed. The skull or the shaven head should be so placed and held that a line passing from the alveolar process of the superior maxilla just at the insertion of the teeth, and through the lowest part of the occipital bone, shall be horizontal. The greatest care should be used to determine this line—the alveolo-condyloid line or plane of Broca,—for it is upon it that all other projections and measurements are based. In the annexed figure the skull is represented as resting upon the alveolo-condyloid plane, 1 – 1.

Next, from the alveolo-condyloid line a vertical line, or one exactly perpendicular to the first, is drawn through the external auditory meatus. At the top of the head this line—the auriculo-bregmatic line,—A–A, indicates the bregma or true vertex, which is an important landmark, one which should be traced with ink or carmine upon the shaven scalp. Upon the top of the head or skull an imaginary horizontal line, 4-4, parallel with the alveolo-condyloid plane, is projected, and upon this line, 4-4, we measure backward 50 mm. and draw a second vertical line, B-B,

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* Written for Gross' System of Surgery. Reprinted from the *Archives of Medicine*, Vol. viii, No. 3, December, 1882.

† See a recent contribution to the subject by Prof. H. B. Sands, "The Question of Trephining in Injuries of the Head." (N.Y. *Medical Journal*, April 21st, 1883.)

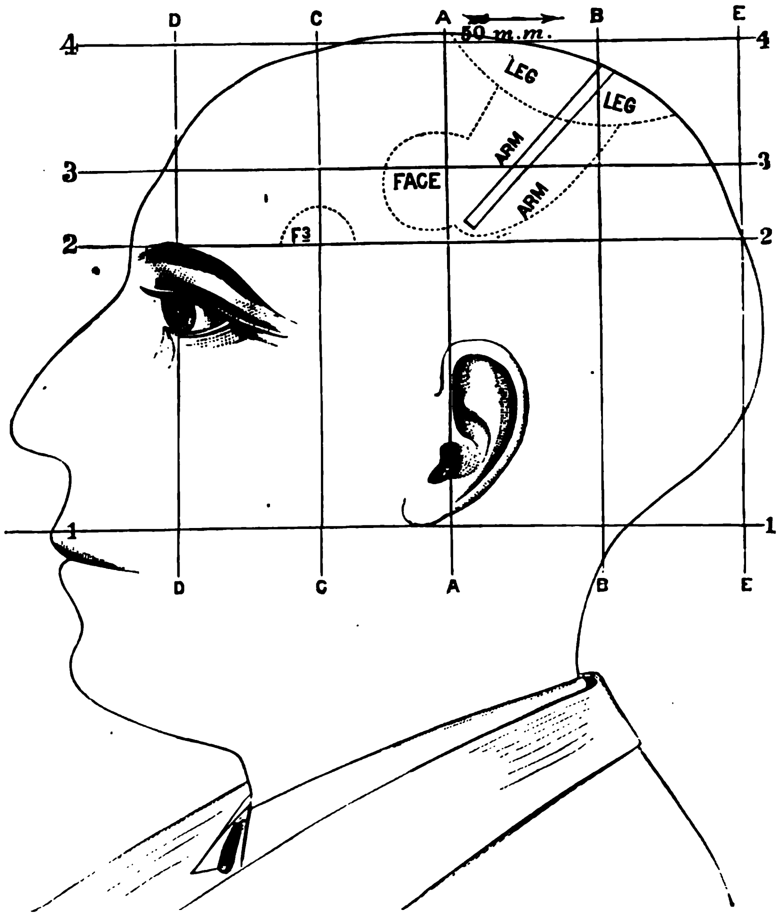

Fig. 1.
Topographical lines applied to the external contour of the head.

parallel to the auriculo-bregmatic line. At the place where this line strikes the convexity of the head is the Rolandic point, R, under which, in average heads, lie the upper or posterior extremity of the fissure of Rolando, the upper ends of the ascending frontal and ascending parietal convolutions, and, within the longitudinal fissure, the paracentral lobule; in other words, the upper part of the motor area of the cerebrum, that which quite probably controls the volitional movements of the legs. The Rolandic point, thus determined, should, on a living subject for operative purposes, be properly marked upon the shaven scalp.

A third horizontal line is next to be drawn from the external angular process of the frontal bone, parallel with the alveolo-condyloid plane. This line, 2–2—which may be called the fronto-lambdoidal, as its posterior extremity will usually pass at or near the upper angle of the lambdoidal suture—serves the purpose of determining the situation of some important parts. In the first place the line 2–2, passes about 5 mm. above the upper border of the squamous suture, and under this line, mostly parallel to it, are the anterior two-thirds of the fissue of Sylvius. Secondly, at about 5 mm. above and behind the intersection of lines A–A and 2–2 is the inferior extremity of the fissure of Rolando bounded by the ascending frontal and ascending parietal convolutions. In the third place, upon this line, 2–2, at a distance of

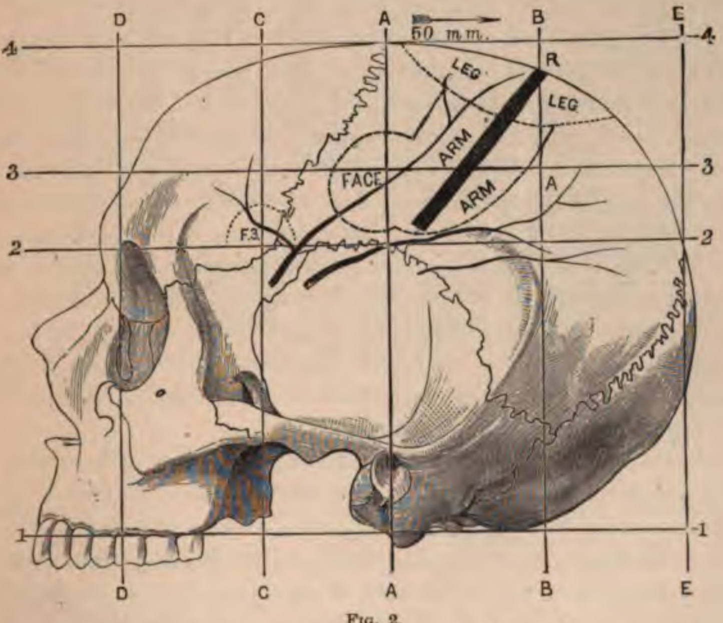

FIG. 2.

Topographical lines applied to Henle's skull. Location of the Rolandic fissure, motor centres, and branches of the middle meningeal artery shown.

18 or 20 mm. behind the external angular process of the frontal bone, is the folded part of the posterior extremity of the third frontal convolution, or Broca's speech-centre, marked F^3 on the diagram.

Having exactly determined and marked the situation of the Rolandic point at the top of the skull, and the inferior termination of the fissure of Rolando above the ear, these two points are connected by a line which is represented darkly drawn upon the diagram. This, the Rolandic line, is the guide for nearly all operations intended for the relief of traumatic spasm or paralysis, since under it and near it lie the so-called motor centres for different parts of the body on the opposite side, as determined by experiments upon monkeys and dogs, and by numerous post-mortem examinations made in cases of tumors and other limited lesions of the brain.

As indicated by the dotted lines on the diagram, the motor zone or centre for the lower extremity of the opposite side lies about the Rolandic point, making an allowance of at least 10 mm. to either side of the median line for the interval between the two hemispheres. It also includes the paracentral lobule within the longitudinal fissure; and we are led to believe, from observation in cases of cerebral tumor, that this part is preëminently the centre for the leg. Below this, reaching quite down to the fronto-lambdoidal line 2–2, is the motor area of the upper extremity. Forward of this, between the auriculo-bregmatic line A–A and the line C–C, is a part of the second frontal convolution, which probably has connections with the facial muscles of the opposite side. Finally, at F^3 is the speech-centre of Broca, which although not now regarded as the only speech-organ, yet plays an important part in the simpler mechanism which produces language-motions.

Other relations of interest are the apex of the sphenoidal or temporal lobe a little beneath the line 2–2, and at about 10 or 12 mm. posterior to the external angular process of the frontal bone; the situation of the occipito-parietal fissure almost immediately under the posterior end of the line 2–2, at its junction with the vertical line E–E, which also indicates the posterior extremity of the cerebrum; the anterior end of the brain is marked off by the vertical line D–D. Furthermore, for certain purposes, the limits of the central ganglia of the brain may be estimated as follows: Their superior limit is indicated by a horizontal line or plane drawn at a level 45 mm. below the vertex, line 3–3 of the diagram; their anterior limit, which corresponds to the head of the nucleus caudatus, is traced by the vertical line C–C; and their posterior limit, or hinder end of the thalamus opticus, by the vertical line B–B. Lastly, it may be stated that the angular gyrus—a part of the cortex which recent autopsies would seem to connect with vision—lies not far from the point of intersection of the lines B–B and 3–3, at the point marked A on the diagram. This, in the living subject, is a little below and behind the parietal eminence.

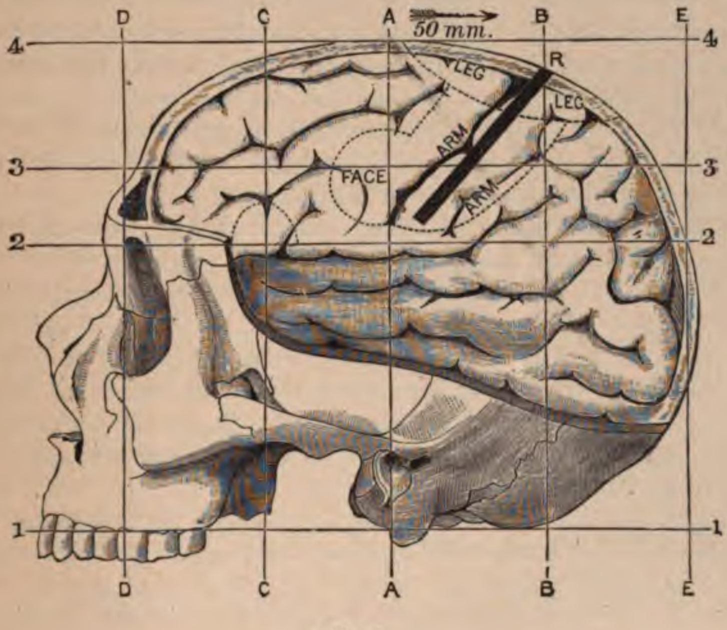

FIG. 3.

Topographical lines and landmarks projected on the convolutions, the apparent non-conformity of the Rolandic line and fissure being due to perspective. Henle's skull.

The location of the middle meningeal artery, which so often furnishes the blood which compresses the brain after various injuries of the head, is surgically considered of great importance. The course of the two principal branches of the artery is approximately indicated upon Fig. 2 by the branching lines drawn on the anterior, inferior angle of the parietal bone.

In the living subject, the main trunk of the artery would be found under the horizontal line 2-2, at a point a little posterior to the speech-centre, about 30 mm. behind the external angular process of the frontal bone, and in front of the beginning of the fissure of Sylvius. It passes obliquely upward and backward almost immediately over the whole of the ascending frontal convolution, from 5 to 10 mm. in front of the Rolandic line. The inferior branch of the artery is nearly horizontal, and almost exactly overlies the fissure of Sylvius.

Upon the shaven head of a patient, seated in a chair or lying in bed, the principal landmarks and relations above defined can be mapped out with a great approximation to accuracy by the use of two rulers, or even by one, to mark the alveolo-condyloid plane, and a card-board cut so as to stand astride the skull in the auriculo-bregmatic vertical. A light wooden apparatus could be easily made to indicate these two lines, while the remaining measurements could be taken with a tape, and the points marked with carmine or black ink.

The practical utility of these anatomical data depends upon an acceptance of modern physiological teaching upon the subject of the functions of the brain. The experimental and pathological evidence now accumulated in favor of the connections of the "centres" marked on the diagram and certain peripheral parts, and between the whole of the motor area, and the whole opposite side of the patient is, as we look at it, convincing, and leads us frequently to a very accurate topographical diagnosis in medical cases.

The following operations may be referred to as illustrative of the utility of the laws of cerebral localization and of cranio-cerebral pathology:

Broca, in 1871, in a case in which aphasia and paralysis followed a severe lacerated scalp wound, trephined over the left third frontal convolution, or speech-centre, found pus, and slightly relieved his patient.

Lucas-Championnière, in 1874, trephined a man in whom coma, partial right hemiplegia, convulsions, and, as shown during convalescence, aphasia resulted immediately from a slight cranial injury. There was only a slight scalp scar to guide him, but he came in contact with splinters and blood from a fracture existing below the point of apparent injury, and saved his patient.

Hueter, of Greifswald, in 1879, in a somewhat similar case, trephined the skull, ligated the middle meningeal artery, and cured his patient. In another case in 1870 he was equally successful.

Courvoisier trephined, in 1878, a child two and a half years old, who, after an insignificant wound, in the left temporal region, had right hemiplegia, coma, and palsy of the left side of the face. He found a fissured fracture, and pus outside the dura mater as well as a large quantity under it. The operation was followed by recovery with weakness of the right side.

Dr. R. F. Weir, of New York, in 1882, operated at Bellevue Hospital, in a case in which coma and slight hemiplegia existed, the patient being a man who had received a blow on the head. There was no very evident external injury, but guided by the various data of cerebral localization, and proceeding according to the rules of cranio-cerebral topography, the trephine was applied, and a small clot found between the brain and dura mater. On incising the latter the brain was seen to be extensively disorganized, and the seat of copious hemorrhage, which was checked by torsion. Although the symptoms were relieved by the operation, death occurred within a few days. For an account of Dr. Sands' case see N. Y. *Medical Journal* April 21st, 1883.

Up to the present time, so far as we know, there are only four cases in which the rules of cranio-cerebral topography have been applied, from measurements actually made prior to operation. These cases occurred in the practice, respectively, of Broca, Lucas-Championnière, Weir and Sands. There are probably other examples, but we are unable to particularize them. However this may be, there is a large number of recorded instances of relatively or absolutely successful operations performed after cranial injuries, for immediate as well as for secondary effects, without measurements.

The indications for trephining after cranial injuries for the relief of symptoms of cerebral irritation, compression, or disorganization, may be provisionally stated as follows :

1. When aphasia supervenes immediately or within a few days or weeks after an injury to the anterior portion of the head on the left side. It is extremely probable, in the first

case, that a clot or bony spicule will be found compressing the speech-centre; and in the second, that an abscess has formed either in the same part or close to it.

2. When simple hemiplegia, or hemiplegia with hemispasm, follows an injury, however slight, in the temporo-parietal region. If the spasm or paralysis be limited to one limb or to the face, the indication to operate is even stronger. Even if the injury be not directly over the motor area, the surgeon is justified in such a case in exploring that area.

3. In conditions of coma after cranial injuries, sometimes without external wound, in which meningeal hemorrhage is the cause of death, the discovery of slight hemiplegia should call for trephining planned according to the rules above laid down, as in Weir's case. A latent hemiplegic state may be discovered, at least in some cases, by an increase of peripheral temperature, as of the fingers or toes, on one side, and by the presence of congestion or of an erythematous blush on one buttock.

4. In the very rare cases in which the paralytic phenomena are found on the same side of the body as the cranial injury, it might be proper to trephine on the opposite side of the skull in search of hemorrhage or fracture, the result of contre-coup.

5. In chronic epilepsy after traumatism of the head, the indication for operation is present, but it is not a specific indication, connected with the subject under consideration. A lesion of any part of the skull may be a cause of epileptic attacks, irrespective of the motor centres.

Contra-indications to trephining may be thus enumerated:

1. Whenever in apparently favorable cases there are signs indicative of lesion of the base of the brain, such as palsy of several cranial nerves, neuro-retinitis, or Cheyne-Stokes respiration.

2. When hemiplegia is accompanied by great anæsthesia, rendering it probable that the lesion is beyond the motor area, deeper, and farther back.

It should be understood that these indications and contra-indications are formulated from the stand-point of the neurologist. The surgeon, upon general grounds, will doubtless often modify them, and add others.

THE TREATMENT OF MILD CASES OF MELANCHOLIA AT HOME.*

GENTLEMEN: The little patient now before you is the subject of a very rare disease—Intermittent Melancholia. He has been under my observation and treatment for about fifteen months. The history of the case, condensed from numerous notes made during that period, is as follows:

Agie Van R——, aged 11 years, is the child of poor parents who have not had insanity or other nervous disease; they are simply ill-nourished and small. Agie himself enjoyed good health until, in the winter of 1873–4, he injured his head by falling backward from a stoop. The scalp was cut through at a point midway between the right ear and the occipital protuberance. The blow was severe enough to produce loss of consciousness lasting several minutes. No symptoms followed this injury, and its occurrence was not remembered by the mother until long after the mental affection had manifested itself. At the time of the patient's first appearance at my clinic, Dec. 19th, 1874, the following notes were made in the clinic record: "Was perfectly well to July 12th. Had been out in the sun; fell asleep, and slept nearly half the time for two days. He was out of his head; restless; singing senseless songs; eating everything; was cross; recognized everybody; complained of headache; could not walk straight. In two weeks became entirely well. After an interval of two weeks, he had another attack in which he was sleepy and out of his mind. Probable hallucinations of vision in this second attack. For some time had such attacks regularly on the fourteenth day, lasting fourteen days. Agie states that in attack before the last (October) he thought he saw bears and thieves entering the room. Last month was treated by Dr. Woolsey Johnson, in Professor A. Clark's clinic, with quinia, obtaining an interval of four weeks. Present attack began on Dec. 13th. Is drowsy, restless, and wants to lie down; many parts of the body are the seat of twitching. Is dull, and speaks slowly when questioned; denies headache; is cross; lies in his mother's lap; pupils normal; sulphate of quinia to be continued. The attack terminated Dec. 24th, and he was then given .001 grain strychnia twice a day, and the mother was directed to apply the wet-sheet. This treatment did not prevent the return of attacks, in January and February, 1875.

"February 8th.—Was well up to yesterday noon, then was rather low. At supper, "fainted dead away" (fit?) and vomited afterward. To-day, looks anxious and depressed; eyes have lost brightness; he slept, but has been singing this morning. Pulse, 90. Ordered bromide of ammonium, .30 and bromide of potassium, .60 every four hours. Feb. 10th.—Has a depressed, sad look; is restless; whistling, chuckling, mimicking, singing. Restless last night. Fundus of eyes normal. Only complaint is of pain in right hinder part of head, and some dizziness. Feb. 15th, eighth day of attack.—Is depressed, noisy, and ill-behaved; pulse, 90. Ordered stop bromides, and take strychnia again, and a teaspoonful of whiskey, several times a day. Feb. 22d.—About well; wants go to play. March 9th.—Melancholia appeared again yesterday; is depressed, and looks pale; not noisy. Pressure on cicatrix in right occipital region, causes a bad feeling in stomach. March 10th.—Patient is etherized, and an incision is made through the scar down to the bone, and a pea put in. Cicatrix is found whitish and tough, adherent to thickened periosteum. Father states that in fainting fit of February 7th, the boy's face twitched, and he lost consciousness wholly. This statement is to be received with much allowance. March 11th.—No worse; still dull and anxious-looking; not noisy. Dressing of wound, while not causing much pain, produces bad feeling in stomach; faintness; at first flushing of the face, then pallor; no twitching or loss of consciousness. The strychnia was stopped a few days ago and the bromides resumed, with whiskey. March 19th.—Coming out of an attack; wound is still open (pea dressing), and a pressure on it is felt in the stomach. March 22d.—Since yesterday morning has been bright and quite himself. Dressing of sore on head produces pallor, sweating, temporary loss of consciousness. Takes iodide of potassium, 1.20, three times a day. April 5th.—On the 3d, awoke irritable and queer; interval a little less than fortnight; yesterday, slept much, ate a great deal, whistled, and sang. General dread without hallucinations; will not go into room alone. Sore is allowed to close up. Ordered bromides of ammonium and potassium, .30 of former, .60 of latter, thrice a day, a double dose at bed-time. Pulse weak and irregular, about 75; tongue clean."

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* Reprinted from A Series of American Clinical Lectures, vol. ii. no. iii, 1876.

During an attack beginning May 3d, the bromide treatment was abandoned and opium given in doses of .015, four times a day, to be gradually increased to six and seven times, if no drowsiness appeared. "May 7th.—Yesterday, was noisy at times; to-day, is quiet and respectful in my office. This morning asked for newspaper, and read the news to his mother; when he is well he does not care to read a paper. Continue the opium, .015, six or seven times, food and stimulants; pulse, 75. May 13th.—Is much better; dressed himself, walked out alone yesterday. Curiously, he has slept less in the day-time since taking opium; yesterday, was not at all drowsy. To have opium .03, four times a day. May 16th.—Perfectly well; attack lasted only eleven days, and was very light. Continue opium, food; cod-liver oil in one dose. May 27.—Remains well. Continue treatment. June 14th.—Has been well for nearly five weeks; looks and feels well; has taken .12 of opium a day, regularly, and began to feel its soporific effect only on the 10th and since. Lately has had no cod oil. To resume oil and take less opium, .03, thrice a day for a week, then twice a day. June 17th.—Was well, up to 1 P.M., yesterday. At 2 P.M. was found by his mother sleepy and taciturn; slept all the evening. No physical or moral cause of attack can be discovered. To-day has usual depressed, anxious look, is not noisy, has no hallucinations; tongue is clean; pulse, 93. Ordered opium, .03, five and six times a day. June 19th.—Not as noisy as in previous attacks. No effect from pills, though he takes seven a day. July 17th.—Has been well for three weeks and three days." I made no note of the case during July and August. "Oct. 19th.—Was well eight weeks yesterday. To-day, mild depression; is not noisy. A fortnight ago, took opium for a week in small doses. Has had cod-oil more or less regularly. To have opium .03 five or seven times a day, according to effect. Oct. 27th, tenth day of attack.—Is brighter and better; sleepy from opium. In this attack was not at all noisy; wept at times. Ordered continue two opium pills a day; also strychnia .001 in Horsford's acid phosphates."

During the later autumn and during the last two months, Agie has had long intervals of health, ranging from nine to five weeks, and has had but three attacks. He is now emerging from one. During January, he took fluid extract of ergot, 2.gm., four times a day, and last month Thompson's solution of phosphorus 2.gm., thrice a day. Cod-liver oil has not been used since the new year. On the whole, very great progress has been made in the case, especially if we take into consideration the bad surroundings in which the little fellow has been placed: his insufficient food, and various depressing moral influences in his home. Many months ago I urged his mother to place him in an asylum, but she preferred to run the chances attending a treatment at home. It is remarkable and gratifying that, in spite of such numerous attacks of melancholia, no dementia has developed.

In connection with this case, I would ask your attention to several considerations of ætiology and treatment, though, before doing this, I ought to present a summary of the symptoms exhibited by our patient. Naturally, and when well, he is a very bright, cheerful, and intelligent little fellow; with brilliant eyes, red cheeks, and strong lines of character in his face. His deportment is unusually good, and his politeness remarkable for his station in life. He is, I am told, very affectionate, and attentive to his mother and sisters. During an attack, he is entirely metamorphosed. His face is drawn, and sad; the light has left his eyes, and the color almost departed from his cheeks; his attitude is relaxed and careless, his dress is disordered, his hair uncared for. His good manners have vanished; he keeps his hat on in my office, asks for fruit while waiting in my dining-room, whistles, kicks the furniture, answers questions reluctantly and in a cross way. At home, he does not stir from a corner or chair, will not rise from the bed, or dress himself, unless forced to; is ravenous and noisy. He has several times had hallucinations of sight, never of hearing. He has (not in every attack) complained of pain or dizziness in the head. Once he fainted (twitched?) at the beginning of an attack. His digestive organs have always been in good order. He has been both drowsy and sleepless in different attacks. I took his temperature many times, and found no deviation from the normal standard; the pulse has ranged from 68 to 100, and I was never able to make out a regular rise in its frequency during attacks; though, on the whole, it has then seemed to range higher. Pallor and some emaciation were present, last year. I caused him to be closely watched for epileptiform seizures, without result.

As regards the mode of production of the attacks, I have thought of several causes. When the boy was first brought to me, I at once investigated the question of self-abuse, or genital irritation, and obtained a negative result; the organs were found healthy, and the boy repeatedly declared his freedom from the evil habit referred to. It then occurred to me that the attacks of mèlancholia might be the result of slight or unobserved epileptiform seizures; and, to elucidate this question, I caused Agie's parents to watch him closely, and made strict inquiries into his past life. As seen in the history of the case, only once was anything observed which might pass for a fit, on February 7, 1875, when he "fainted dead away," and his father, an unreliable witness, thought that he twitched. Against the epileptiform nature of the disease we have, furthermore, the failure of treatment by the compound bromide solution given in large doses from the beginning of February to the end of April. When, during the month of February, I learned of the injury to the head, it occurred to me that a morbid state of the meninges under the injured bone might be the cause of the symptoms. I accordingly performed an operation, and kept a sore running for weeks, at the seat of injury, but without relief. I was not prepared to advise trephining until a further trial of medicines had been made. The opium treatment appears to have had the best effect, when conjoined with cod-liver oil and other tonics. The approximate success obtained during the last six months by these means would point to mal-nutrition of the brain, as a cause of the melancholia: a pathological state not rare in the melancholia of adults.*

Another case which has been treated at the clinic, and with greater success than the preceding, is that of Mary L., a married woman, aged 25 years, brought here August 29, 1874. "Has been married about four years, and has borne two children, nursing them both. About two and a half years ago, after the birth of the first child, she experienced a choking sensation in her throat; felt as if she wanted to cry. This disappeared after a while, but reappeared after the birth of the second child, a few months ago, the attacks of choking being preceded by a sense of cold in soles of feet and at wrists, accompanied by nausea, and by desire to weep. Is low-spirited and imagines that she is going to be sick, or that some disaster is to happen. Has nursed child constantly and freely, and has besides had sexual intercourse very often. Is thin, pale, and weak. Facies very despondent, patient is convinced that she cannot get well. Confesses that for many weeks she has had fearful impulses, to go and drown herself in the river, and to kill her children. The latter impulse surges up frequently within her, and she has had to fight hard to resist it. She has made it a rule to lock up all knives or other sharp instruments in her rooms; and lately has, by her express wish, been closely watched by a woman in the day, and by her husband in the evening. Denies most positively having had any hallucination of sight or hearing. Has been careless of her home, of her dress, and person; has lost interest in everything; is extremely depressed, and often weeps. Reasons well upon all matters which usually are talked of by a woman in her station of life. Remembers everything well. Is inert, and indisposed to take any exercise. Often very wakeful at night.

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* In a few months after the publication of this lecture, Agie was quite cured, found employment, and has remained well.—[E. C. S.]

I recognized this as a case of melancholia without delusions, but with strong morbid impulses. There had also been an hysterical element in the case. The causes of the exhaustion of physical and mental vigor were evidently prolonged lactation and too great sexual indulgence. I at once began a treatment based upon this view of the pathogeny of the case, enforcing weaning of the child, separation of the husband and wife at night, and ordered food and tonics. She was given ale at night, meat and other nutritious food thrice a day; was made to walk every day. In order to secure sleep, I prescribed a pill containing .03 of extract of cannabis indica and .015 of powdered opium, to be taken three or four times a day, according to the effect produced. The patient was in the habit of rising at night to answer any cry of her infant child, and this was prevented by having a woman take entire charge of the child. At all times Mrs. L. was to be under guard to aid her in resisting the impulses described above. She was to stay a part of the day in her husband's store, and do what little she could to help him. Her husband and friends were strictly enjoined to do or say nothing tending to depress her or excite her emotions. I told the patient in the most positive terms that she would recover in a few weeks, and reiterated this assurance at every visit, though she often shook her head and said it was no use. Cod-liver oil was added to the treatment in October, and the number of opium and cannabis pills was reduced, at first, to two a day, later to one at bedtime. On October 5th, I gave her, after meals, a pill containing extract of nux vomica .015, zinc phosphide .01 gm. and zinc oxide .06. This was discontinued on October 23d, and a mixture substituted, each drachm dose of which contained .003 of strychnia, and 2. of Horsford's acid phosphates, with 2. of syrup of orange flowers. At first, Mrs. L. took three doses; later, in November, four doses a day, continuing the cod-liver oil and ale. The last prescription which I wrote for this patient was on November 24th, 1874, when I gave her .30 of pyrophosphate of iron three times a day. At that time she was perfectly well. The improvement had appeared very soon after the patient began to rest at night and ceased exhausting herself. She gained flesh and color rapidly, and, *pari passu* with the physical gain, came a mental improvement, greater tranquillity of mind, a little hope of recovery, interest in matters of every-day life, and ability to banish every thought of injuring others. I urged the patient to exert her volition to the utmost, when such ideas arose in her mind, and to seek relief from them also in occupation. Corresponding with these changes in the bodily and moral state of the patient, a degree of healthy coquetry made its appearance, as shown in the arrangement of hair and dress.

A case, similar in many respects to the above, occurred in my private practice during the past autumn.

Mrs. Lev., aged 27, came to my office, September 22d, 1875. She had been married six years, and had had four children in that time, not nursing any of the children, and not having any complications in labor. Last confinement occurred in January last, and after it she was not well; her children were sick, and she obtained but little sleep for four months. Had pain at top of head and in left side of abdomen, nausea, and pain at the pit of the stomach. A physician in Mobile made an examination and found the uterus "ulcerated." Was frequently purged, and received local treatment, with relief to nausea, pain in the head and abdomen. She, however, grew weak under this treatment. In the last few months, chief symptoms have been mental and moral; the patient has become despondent, wished for solitude, expressed her inability to do anything, and her conviction that she is crazy and will not recover. Her sleep has been unrefreshing, and she has not seemed to gain any strength from food. Lately, the negative mental state has become very pronounced, and patient must be urged to rise, to dress, to eat, to go out. She is apprehensive of softening of the brain, etc., and states that her head feels dull and stopped up; she feels childish, foolish, and irritable. Shakes her head incredulously when I tell her that she is sure to get well. The memory is a little impaired for recent events; there is no incoherency in speech; no delusion; and patient denies having had any hallucinations. She is conscious of the feebleness of her will, and of the torpid, helpless state in which she is. Complains of no pain, but has at times creeping sensations over the whole body; sometimes is a little dizzy, and has a feeling of pressure at the back of the head. Has a tender point in mid-dorsal region, and before treatment of uterine disease had one in the lumbar spine. The menses have appeared irregularly at five or eight weeks' interval, have been copious, and have caused weakness. There are now no symptoms of uterine disease. The fundus of the eye is normal; the face pale, and the patient's eyes underscored with black. She has become emaciated, and is comparatively weak. This patient often said that she had rather die than be in the state she was, but never had any strong suicidal impulse, and never any desire to injure any one. The prominent psychological state was the negative condition, in which she found herself without reaction to the usual stimuli of life. She said that she could not rise in the morning, could not dress, could not take medicine, could not walk out. "I am worse than a child," was her oft-repeated complaint. I made the diagnosis of physical prostration and melancholia sine delirio, and encouraged her friends to believe that she was to recover in a few months. Toward the patient I adopted a more positive tone, and told her that she would get well surely, positively; and that before Christmas she would be a healthy woman. This statement I repeated as a part of the treatment at every interview.

I prescribed a moral and a medicinal treatment. In the first place I instructed the friends of Mrs. Lev. to watch her constantly, because, though there was no tendency to suicide then present, such a disposition might be suddenly developed and an attempt made to gratify it. The patient was to be aided by the will of others in all things; she was to be *made* to rise at a certain hour, if necessary, by the stern means of taking away her bed-covers. She was to be helped and expedited in the operation of dressing, and brought down-stairs in time to breakfast with the family. The same punctuality was to be enforced in other things. Regularly, also, she was to be taken out walking, twice a day at least; and, after a while, when better, I directed that she was to be obliged to take care of her room. Now, gentlemen, this was not tyranny, as one unacquainted with the subject might think, but a kind assistance to the patient. She was glad that the will of others was interposed to help her out of the negative state into which she had drifted. Of course, in carrying out this plan, no physical violence was to be used. I told the friends to throw the odium of enforcing disagreeable things upon my shoulders, by saying: "The doctor says it *must* be done."—"You promised the doctor that you would do it," etc. I furthermore enjoined them never to deceive the patient in order to make her do anything or to gain her good-will. This means is one which I believe should very seldom be resorted to, in mild cases of insanity. In addition, Mrs. Lev. was to be amused moderately, invited to play cards or backgammon, and later, when stronger, was to be taken to matinées at the theatres.

In the second place, I ordered the following medicinal treatment: The patient to go to bed at 10 o'clock, after drinking the better part of a bottle of porter. Her three meals were to be substantial; meats, eggs, etc. With her dinner she was to take a pint bottle of porter. Cod-liver oil was prescribed in doses of 4. after each, meal to be slowly increased to 15.; and I gave .001 of phosphorus, in the shape of 2. of Thompson's alcoholic solution, thrice a day. The galvanic current was applied to the head a few times, with the effect, apparently, of relieving the dysæsthesia of which she complained. October 5th.—Not much improved in mental condition; it is difficult to enforce rules of life prescribed; patient still very obstinate and quite hopeless. The cod-liver oil and phosphorus are given together in an emulsion, according to the following formula:

℞. Vitelli ovor,
Ol. morrhuæ, āā, 120.
Vini xerici, 60.
Sol. phosph. (Thompson's),
Syrup. simpl., āā, 15.
Aq. amygdal. amar., 120.
S. 30. after each meal.

On October 11th there was added to the treatment a pill of cannabis and opium, .02 each, to be taken at bed-time with the porter. October 29th.—Great improvement, physical and mental; patient begins to believe that she will recover; shows more spontaneity; she shows disgust for the emulsion of phosphorus and oil, and is ordered Caswell's oil with hypophosphites of lime and soda, 8., after meals, to be increased. Porter, twice a day, as before; opium and cannabis at bed-time. In the early part of December, Mrs. Lev. was very much better, almost herself in fact. I directed that the opium and cannabis pills, and the cod-liver oil be no longer given, and prescribed the following tonic and stimulating pills:

℞. Ext. cannabis, 0.20.
Ferri sulph., exsiccat.,
Sodæ carbonat. āā, 8.
M.
In capsul. no. xxx., divid.
S. Two after each meal.

By Christmas-time recovery was quite complete. The patient became anxious to go back home, to care for her house and children, and was only prevented from going by being led to expect her husband from week to week. Until his arrival, in February, 1876, Mrs. Lev. amused herself much, and though not taking medicine, observed the hygienic and dietetic rules I had laid down for her guidance. Her husband's arrival was the occasion of a severe test of her recovery, and he was obliged to tell her that, during the early winter, one of her children had died. This news, gently imparted, provoked an outburst of passionate sorrow, but no relapse.

Another case in my private practice, illustrating the points I wish to lay before you, was that of Mr. M., aged 28 years, married, and a wine merchant by occupation. He had been a healthy and temperate man, whose business, though prosperous, had pressed heavily upon him. From being a travelling salesman, he had become partner, and he felt the responsibility acutely.

During the winter of 1873–74, marked physical fatigue and slight chronic lowness of spirits manifested themselves. In February, 1874, Mr. M. slept less well, waking about early dawn and tossing uneasily about until the hour of rising. In March he became more depressed, ceased taking part in social games, needed spurring about everything, and began to speak of the bad way his business was in, and hinted at approaching ruin and beggary. In point of fact, this was the beginning of a delusion, as his affairs were in a fairly prosperous state. In April, after taking quinia pills, he began to speak about a stoppage in his bowels, and frequently remarked that he ought to die.

When I first saw Mr. M., in April, 1874, he was sitting in a chair, wearing a most melancholy expression, in strange contrast with his ruddy cheeks and general appearance of good health. He only half rose to greet me, and gave me a lifeless hand. He was in good flesh, his pulse strong, and not above 80. His tongue was much coated and his breath very foul. His frame of mind was that of despair, yet he was not emotional. "You can't do anything for me, doctor," he said. "My bowels are stopped and nothing will go through me; I shall choke with all the food they force me to take; my business is wholly ruined; we are beggars now; I can't go to business, I can't exert myself." He would put his hand on his abdomen and say, in the most despairing way: "There it is, doctor; you can't put anything through me." He had been sleeping less and less well of late, in spite of bromide of potassium which had been given him by a friend, in considerable doses, for weeks. No hallucinations.

I made the diagnosis of melancholia with delusions, and informed the wife that success in treatment at home depended upon her vigilance and our success in overcoming his objection to food. In case he positively refused to eat, I should be in favor of his immediate transfer to an institution.

I administered croton-oil pills, broken up in tea, with the result of giving him many free movements; but the sight of the evacuations and our arguments did not dispel the delusion that his bowels were stopped up. He continued for weeks to entertain this notion, and to protest against being fed. He was like many insane persons, reasoning correctly upon a false premise. I was fearful that he might draw from his other belief, viz.: that he and his were beggars, the conclusion that it would be desirable and proper to kill his wife or himself. Consequently I enjoined upon his wife to remove from the room anything which might serve as an instrument of injury, to have the windows nailed fast, and to watch him incessantly. I directed that nutritious liquid food be given to him frequently; beef tea, chicken broth, milk punch and egg-nog. In reality, he was supported chiefly upon milk and brandy, taking as much as eight and ten ounces of the latter per diem for two weeks and more; when, as he began to eat more, the amount was lessened. The bromide of potassium was discontinued, and I prescribed chloral to be given in case the stimulant did not make him sleep. I believe that few, if any, doses of chloral were necessary. As a tonic, and with the view of counteracting the depressing effects of the bromide of potassium which had been taken during so many weeks (no eruption produced), he was ordered:

℞. Strychniæ, 0.06.
Acid. phos. dil., 30.
Syr. aurant. cort., 90.
M.
S. A teaspoonful three times a day.

Fortunately Mr. M., although protesting against being given food, which must accumulate and choke him, yet remained good-natured, and made no physical opposition when his wife fed him and told him that it was the doctor's positive orders that he must eat. After a fortnight the refusal became less strong and gradually disappeared. He was taken out to walk once or twice a day, helped in dressing himself, and, when better, made to go down into the parlor and see friends. He often, during April and May, assured me that I would never be paid, as he was a beggar, etc. After the 1st of June, the patient rapidly improved, sleeping and eating well, and using much less stimulant. On June 13th I substituted for the strychnia mixture the following:

℞. Ext. nucis vom., 0.60.
Pulvis rhei,
Ext. cannabis, āā, 1.
Quiniæ sulph., 2.
M.
In pil., no. xxx., divid.
S. One before each meal.

About the middle of June, Mr. M. went to his father's place on Long Island, his wife accompanying him and watching him without his being aware of it. He worked about the house, bathed in the surf (never alone), and steadily improved. During the early part of July he referred to the locked state of his bowels for the last time. The last wrong notion to disappear was that about his business. In September and October he was well, only he feared to go to business because of mortified pride. He was afraid of remarks about his having been crazy. He had no more delusions, but his will was very feeble (it had never been strong), and it needed all his wife's exhortations and my own words of cheer to induce him to start life anew. He remembered all that had taken place, and was ashamed.

In these four cases, gentlemen, we find the chief symptoms of the melancholic state, or the condition of depression.

A. Psychic pain.—This element, difficult to define, was present in all the cases. It was indicated by words, and, better still, by the expression of the face, and the attitude of the body. The patient feels low-spirited, is without hope, the world appears as if a black pall had been thrown over it, friends are careless or have become enemies, everything goes wrong. This condition of the mind is one with which nearly all of us are acquainted in a milder degree, constituting what is popularly known as a "fit of the blues." In many persons the "blues" amount to a short attack of melancholia with positive delusions, lasting one or two days. Satisfaction with the past, contentment with the present, and hope for the future, as well as all energy and power of enjoyment vanish when the fit begins. And, I believe, from the experience of my friends, and from my own, that such attacks of transient melancholia are often the result of overwork or of mental strain. Some individuals will have such a fit quite surely after a month or two of hard professional work, just as another will close such a period by an attack of sick-headache. A day of desperate brooding succeeded by a night of unusually good sleep, brings the attack to a close, and the subject awakes brighter and more energetic than he was for days before the storm; again, just as occurs after a sick-headache.

B. The negative state.—Whether from absorption in his mental wretchedness, or because of the influence of dominating or terrifying delusions, the patient shows no spontaneity in action; he fails to react normally (often does not react at all) to external stimuli or to the incitations which may arise within him as results of preserved intellection. A mode of expression of this negative state is the unwillingness of the patient to act, and his liking to sit or stand still in one position for minutes, or hours, or days. Many melancholic patients (while still preserving reasoning capacity) will never rise from the bed, or dress, or eat, or walk, unless made to do so by an impulse from without. This negative state was present in Cases I., III., and IV., and faintly developed in Case II.

C. Impairment of volition.—This was more or less marked in all the cases. In Case II., the patient, while unable to overcome her depression and inertia, was yet able to control the frightful impulses to murder her children. Yet she felt that her feeble volition was not to be depended upon, and took the precautions to have some one with her all the time, and to lock up all cutting instruments. No one could say when the impulses might become irresistible. In many subjects, volition seems to be utterly absent (or unused) for positive purposes, for inciting to externally manifest acts, but appears excessively developed in a negative or opposing way, as in refusing food or objecting to taking exercise. In mild cases, no such obstinacy exists, and patients yield to positive commands with greater or less readiness.

D. Morbid impulses.—These were, I think, present in all the cases. The young lad whose case I first related was led by these impulses to do many disagreeable things, whistling, crowing, stamping, kicking, etc. He was never aggressive or wicked, never stole or attempted to set fire to anything. Yet, I take it, that he was a subject well-disposed to insane thieving or incendiarism (technically known as kleptomania and pyromania). In Case II., the impulses were constant and copious, and they were of a murderous type, tending to cause homicide. The patient's depressed mind was agonized by the dread that she might fail to resist the impulse. Suicide might logically have resulted from this condition, the patient having reasoning power left to prefer killing herself to injuring her children. In Cases III. and IV., the morbid impulses were weak or concealed.

E. Hallucinations.—We understand by hallucination the occurrence of a false sensorial impression. The boy thought he saw thieves in a room, but there were none to see. A patient will hear voices when there are none sounding, or will smell odors when no one else can perceive them. Another patient will declare that certain sensations occur inside of him or upon his skin, when a careful examination shows no reason for believing that any such sensations can exist. I would remind you that a person can have hallucinations of any of the senses, and yet not be insane; the difference in this respect between the sane and the insane being that the mentally healthy mind corrects the false impression by the exercise of reasoning, or by means of tests applied by the other senses. For example, a person having an hallucination of sight (seeing an animal or a man) will make sure by touch or by reasoning that no one *is* there. Again, one who has lost a limb by amputation, may have an hallucination of the sense of touch, leading him to believe that the amputated part is still attached, but he corrects the error at once, even though the impression be very vivid. The insane, on the contrary, accept an impression as true, and the hallucination then constitutes a delusion. Physiologically, a hallucination is to be looked upon as an outward projection of a deep-seated sensation or irritation of the special centres for sight, etc., projected outward into the external world as images, etc. I might take this opportunity of remarking that prolonged and vivid hallucinations of hearing in the insane make the prognosis more unfavorable.

F. Delusions.—In my spring lectures upon insanity, I have proposed to classify delusions into sensorial and notional; or, into those which consist in a firm belief in unreal objects, or sensations (connected with hallucination), and those which consist in a belief in unfounded ideas. For example, in case I. the patient believed that thieves were in the room, and had a sensorial delusion which no amount of reasoning could dispel. In Cases III. and IV., the false beliefs were psychologically different: in the one case, the patient believed that she could not recover, and in the second, that he was ruined and that his family were beggared. Or, a subject will, in melancholia, believe that he is damned, or, in general paralysis, that he is the father of fifty thousand children, eighteen thousand of whom are black, as in a case observed by my friend, Dr. Chas. Langdon, in the Hudson River State Hospital. In some cases, it may be difficult to draw the line between sensorial and notional delusions, on the one hand, and between notional delusions and eccentricity or peculiar belief, on the other hand.

G. Preservation of the mind, or of the memory and the power of reasoning, exists, in most cases of melancholia which are to get well.—In our cases, this preservation was almost complete; in only one case—Case III.—is diminution of memory noted. In all the cases, however, the reasoning powers were torpid, or appeared so, because of the difficulty of observing their operation. It is common, however, for persons recovered from insanity to tell us that they had full use of their powers of observation and reasoning during their illness, but could not make this activity externally apparent. A knowledge of this condition will guide you in your relations with insane patients, leading you to use great kindness (though combined with firmness) and honesty toward them. I would have you know that a harsh word, a blow, a trick, or a satirical remark, will be remembered by nine patients out of ten.

I have occupied so much time in speaking of psychological symptoms that I cannot add much about the physical ones; they are sufficiently detailed in the histories of the cases, and bespeak prostration of the nervous system, anæmia, and especially mal-nutrition of the brain.

As regards the importance of anæmia, as a pathological factor, in these and analogous cases, I would say that it is often overrated and wrongly stated. That the general anæmia (spanæmia) should lead to impairment in the nutrition of all tissues, and of the brain in particular, I admit, with every one. But that chronic diminution in the quantity of the whole mass of blood shall lead to the development of melancholia in a direct manner (*i. e.*, by producing ischæmia of the brain) I consider extremely doubtful. In other words, I hold that rarely, if ever, is chronic anæmia a factor of as great importance as mal-nutrition of the anatomical elements of the brain; a mal-nutrition which may occur while the organ is receiving a normal quantity of normal blood, being brought about in such a case by excessive activity (waste) of the anatomical elements. I entertain analogous objections to the generally received idea that congestion is frequently a leading pathological factor in cerebral diseases, and furnishes the chief indication for medication.

The ætiology of the cases is worth recapitulating, as in it we find indications for treatment. The first case I would separate in this connection, because it is anomalous in its manifestations, and probably many causes (paternal vice, insufficient food, and possibly the action of over-heat) co-operated to bring about the morbid state. Besides, I would not, even now, after months of observation, positively deny the epileptic origin of the oft-recurring attacks.

In Cases II. and III., types of a very numerous class, long-continued drain both upon nerve power and upon the nutritive fluids (too frequent child-bearing, lactation, and excessive sexual indulgence) was the cause. In very many cases, the numerous factors (insufficient food and clothing, alcoholic and sexual excesses, thwarted desires, oppression, anxiety for daily bread, etc.), which go to make up the condition briefly called "misery" in the lower classes, come into play. In Case IV. we see the operation of a moral cause (so-called) upon a healthy though rather feeble-minded individual. He could not stand the advancement and prosperity which his industry and faithfulness in a subordinate position had brought to him. Anxiety, of a purely imaginary kind, overpowered him.

Now, gentlemen, about the management and treatment of such cases. After you have made a diagnosis in a case of insanity, the first question which will come up for consideration between you and the family of the patient will be the momentous one: Shall the patient be treated at home, or shall he be sent to an asylum for the insane? As upon the decision of this question may depend not only the life and reason of the patient, but also the existence of persons around him and the preservation of property, I think it worth while to give you all the aid in my power to lead you to a right decision. In the first place, be prepared for a protest on the part of the family against removal, and assertions that they will never consent to it, that it would kill the patient, etc., and do not let your judgment be influenced by such clamor; the question is one not be decided by considerations of sympathy and affection, but by reason and the light of experience. Three chief points should be studied in this connection; 1st. Are the hallucinations, delusions, and impulses of the patient vivid and strong? 2d. How obstinate is his refusal of food? 3d. How distinct is the tendency to suicide? I am speaking only of cases of melancholia, please remember.

If hallucinations and delusions are strongly marked, the other two states will most probably also be largely developed. The refusal of food, when positive, obliges us to feed patients by means of the stomach tube—a procedure almost impossible in private practice—as the operation should only be done by a medical man. If the suicidal tendency is strongly developed (if the patient fancies, for example, that a celestial voice bids him kill himself to avoid damnation), the watching at home will not be sufficient to prevent the accomplishment of the wish. Patients will wait months for an opportunity to throw themselves out of the window, or to drown themselves. My rule is in such a state to declare that the patient must be placed in an asylum, or that I can have nothing further to do with the case. I can hardly conceive of arrangements which could induce me to take charge of a severe case of melancholia confined in a private house (except in the late, incurable stage in which dementia is present). Another reason for advising the removal of the patient to an asylum, or separating him from his relatives, is when he connects most of his melancholic delusions with the members of the family, or when their presence appears to plunge him in the slough of despair and psychic pain. Besides the considerations named above, there is one which has great weight with the relatives of patients, viz.: that the chances of recovery are much increased by an early removal to an asylum.

If the symptoms enumerated above are not strongly marked—if, in other words, the case is one of mild melancholia—you may, I think, properly undertake to treat it at home.

By what means? In the first place, by kind, firm, and judicious management as described in the cases related in the first part of the lecture. Instruct the relatives and nurses to watch incessantly, to prevent the accomplishment of a concealed plan of suicide. I would have you feel that every depressed patient *may* commit self-destruction. The minutiæ of this watching and care I cannot enter into; they will readily occur to you. Secure a cheerful moral atmosphere for the patient as far as possible; make him eat, go to walk, engage in simple games, pay some attention to social duties. You may use an authoritative tone and manner toward him without failing to be kind and considerate. Let your positiveness be his helpful stay; let your will replace his own which is so feeble.

Many such patients will need "building up," which means the giving of a more nutritious food in larger quantities, together with the extra foods, alcohol and oil. This "building up" would, of course, be in vain, if you did not look after every possible source of drain upon vitality, lactation, menorrhagia, uterine disease, repeated child-bearing, sexual excesses, legitimate and solitary, and put a stop to the one you find active.

As aids to improve nutrition and strength you have a choice among many tonic medicines, including some of the class "restoratives" of Headland, such as hypophosphite of lime and soda, free phosphorus,[*] iron, manganese, arsenic, strychnia, alcohol.

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[*] Free phosphorus may be given in ethereal, alcoholic, or oily solution. One of the best (and my favorite) modes is by Thompson's solution of phosphorus (*The Practitioner*, 1873, II., p. 13, p. 271), the formula for which is as follows:

Phosphorus, gr. 0.40.
Absolute alcohol, 120.
Dissolve and add:
Glycerin, 270.
Alcohol, 15.

Mix the two solutions and, while hot, add essence of peppermint 15.

4. of this solution contains nearly .002 of phosphorus.

It may be given as it is, or with more glycerin, or with cod-liver oil.

In order to produce sleep, I would advise you to use either chloral or opium. The bromide of potassium is generally prescribed, but without much good effect. We do not know that it is a direct hypnotic, and the quietude which its continued (days or weeks) use brings about, is accompanied, I am convinced, by mal-nutrition of the cerebral tissue. Thus, while it may for a time do apparent good by preventing restlessness, it injures the patient by perpetuating a condition of the brain which we believe to be fundamental in the melancholic state. The use of chloral, if not too long continued, is better, and less injurious. We can produce quiet certainly by this drug, and most patients can take it long without showing symptoms of chloral poisoning.

Opium, cannabis indica, and alcohol often make melancholic patients sleep very well; and, besides, they improve the nutrition of the brain, render the circulation in it more active, and thus expedite *cure.* These drugs belong to the classes narcotics and deliriants of Headland: they first, when given in proper doses, produce intoxication, and then depression and sleep. To all of the patients, about whom I have talked to you, I gave alcohol, in some shape or other—porter, milk-punch, etc. All but one took opium and cannabis. These medicines I give in pillular form, pushing the opium up to the point of slight narcosis (which is difficult to produce in these cases), and seldom giving more than .10 cannabis in the twenty-four hours. Or, as has been practiced lately in France, with great success, morphia may be given hypodermically twice a day in gradually increasing doses. This plan, in private practice, is open to the objection that the physician must make too frequent visits.

Galvanization of the spinal cord, and of the cervical sympathetic, has been used in Europe and in this country, in the treatment of melancholia, with apparent success. It is important to apply the current continuously (*i. e.*, without shocks), in order to obtain a good effect. For particulars of the methods recommended, I refer you to Althaus, Medical Electricity, London, 1873, p. 483.

There are many other points in the treatment of mild cases of melancholia at home, which I have not time to dwell upon, such as the regulation of the action of the bowels, sponging with cold water, or the use of cold compresses, the choice and gradation of occupation and amusement each day, etc. Your common sense and medical knowledge, together with a knowledge of the patient's habits, will guide you safely in these matters. I have the greatest faith in the efficacy of amusement and employment in the treatment of insanity in general, and of melancholia more particularly. Reading aloud to the patient, inducing him to play some simple games, backgammon, dominoes, croquet, billiards, taking him to places of interest, and, when convalescent, to minstrels or theatre. All these things will be of service. For women, knitting, sewing, the care of a room, will be proper occupations at home. There comes a time, in convalescence, when travel is a most valuable remedial means, involving, as it does, change of climate, variety of food, exercise, pleasant sights, and association with strangers. In the moral treatment we must aim to displace the painful, depressing ideas which surge in the patient's mind, and try to break up the chain of association between morbid physical conditions and unhealthy mental states.

I would close by warning you that you will need to exercise much patience in the successful management of such cases, that you should make the family understand that the treatment will last months, and that, if you are not faithfully and actively aided by the patient's friends or by nurses in the carrying out the moral treatment, your medication will prove quite useless.

THE CULTIVATION OF SPECIALTIES IN MEDICINE.*

OF the many practical questions which present themselves to the minds of students of medicine, and even more forcibly to the minds of young graduates, few, I take it, are more interesting than the one: "Shall I practice medicine in general, or become a specialist?"

The importance of this question seems to warrant my making it the text of remarks on this occasion, when the Faculty of the College have delegated to me the pleasing duty of bidding you welcome.

The growth of specialism in medicine is quite modern, I might say recent, yet its germ is ancient. For example, in the celebrated medical school of Alexandria, and among Arab or Saracen physicians in the first six hundred years of our era, we find mentioned as special practitioners, surgeons, lithotomists, oculists, and midwives. On the other hand, I doubt not but that more than one of the venerable pillars of our alma mater, the senior professors who are with us this evening, clearly recall the time when there were no specialists in the United States; a time when all practitioners of medicine, somewhat arbitrarily divided into the classes of physicians and surgeons, knew all there was to be known of medical science, and successfully enough practiced in a corresponding general way. Then no one devoted all his energies to the critical study of changes in the human cuticle, or spent hours peering into eyes with a little mirror, and racking his brains over complicated mathematical formulæ to correct nature's failure to produce a perfect eye. No one made it his exclusive business to light up, expose, and more or less barbarously medicate the various cavities and recesses of the human body, and no one (worst of all, I have heard it said) gave up all practice except that in connection with the nervous system. Were those the better days?

In the last thirty years all this has changed. Quite an army

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*Address introductory to the session of 1880–81, at the College of Physicians and Surgeons, New York; delivered October 1, 1880. Reprinted from the *Archives of Medicine*, vol. iv. No. 3, December, 1880.

of specialists has sprung up all over the world; one specialty after another has made formal demands for recognition in the midst of the profession, and in the faculties of medical schools. Indeed, the human body has been so parcelled out to suit the demands of study and practice by specialists and pseudo-specialists that there is probably no room to spare; and the general practitioner is seemingly justified in exclaiming: "Would these specialist neighbors of mine leave me nothing to do?"

I repeat that specialties and specialists have increased remarkably in the last few years, and, planting themselves in large cities, have demanded the exclusive control of such cases of disease as seemed to fall within the limits of their respective branches of practice, and at the same time claiming (often wrongly, I am sure) superior knowledge of such matters.

This rapid growth, the rather loud claims, and the apparent great pecuniary success of specialists, have, naturally enough, roused in the ranks of the profession at large some adverse criticism and opposition. It has become rather fashionable, I suspect, to conveniently ignore the successful diagnosis and practice of specialists, and to pick out and hold up in full view their mistakes and failures. Yet, gentlemen, I appear before you to-night, prepared to maintain that the growth of specialties has been, and is, of the greatest utility to medical science and to the welfare of the public; and, also, that the practice of a specialty is, under certain conditions, perfectly right.

The growth of specialties is justifiable on the ground of its having been a natural and an almost inevitable development.

No ambitious or ingenious physician planned the creation of a special practice, but specialties have slowly risen up in accordance with the demands of the age; an age of unparalleled accumulation of human knowledge and of wonderful fertility in means for the application of such knowledge to practical uses. In this general proposition are included a number of immediate causes of the growth of specialties, and some of these I purpose briefly to review.

1. Early in this century, a considerable number of physicians in Europe, seem to have realized that a life-time of study would barely be sufficient to enable them to become conversant with the enlarging mass of medical knowledge, and that such an universal knowledge, if attained, would not be thorough enough to fit them for universal practice. Besides, the time consumed and

the mental energy employed in this general study were incompatible with original research and progress.

Probably because of such ideas, together with the prompting of progressive genius, we find that certain members of our profession, without becoming special practitioners, began and carried out special studies, and in several instances these special studies have made their authors immortal.

For example, let me name Lænnec, in what we call physical diagnosis ; Bright and Rayer, in diseases of the kidneys ; Bayle and Esquirol, in so-called mental diseases; Abercrombie and Ollivier, in diseases of the brain and spinal cord; Hope and Bouillaud in affections of the heart; Cruveilhier, in pathological anatomy; John Hunter, Bichat, Magendie and Müller in anatomy and physiology.

Each of these great men for years devoted almost all his energy to the cultivation of what then seemed the outlying fields and dark by-ways of the domain of medical science. Had their ambition been, on the contrary, to be walking encyclopedias of medical knowledge, what would we say of them to-day?

2. It is very probable that the methods of thought and manner of work of medical men in the first third of this century were considerably influenced by the development of specialties in general science.

In previous times a few great men in each century had appeared with a master-knowledge of the whole of the science of their day. Such, for example, were Bacon, Linnæus, Buffon, and, to a certain extent, Swedenborg. The birth of the natural sciences in the troubled times of the latter part of the eighteenth century may be looked upon as a sort of revolt against this assumption of universal wisdom by a few, and the beginning of independent, divergent, special work by the many.

If we take up this movement in the first half of our century we see, as examples, chemists busy for years at different branches of their science; some searching by analysis for elementary bodies, or for alkaloids in plants ; others attempting the synthesis of substances ; others yet endeavoring to discover chemical products which can be immediately useful in the arts, etc. We note the development of zoology into a great tree of knowledge whose various branches,—comparative anatomy, ornithology, ichthyology, entomology, paleontology and anthropology,—engage the attention—the special attention—of innumerable observers. Histology, animal and vegetable, has arisen as a separate science; and so has embryology. In other departments we see men devoting themselves for years or for a life-time to the study of light, of electricity, of nebulæ and stars, of climate and weather, etc.

To close this enumeration, let us say that the great scientific progress of the last fifty years has been the result, in greater part, of specialized research. And in the same period the men who, having a vast store of knowledge, have attempted to generalize the labors of specialists are exceedingly few. Perhaps I do not exaggerate when I say that Charles Darwin is the only one whose efforts in this direction have been deemed deserving of universal acknowledgment.

How could medical men, medical scientists, in constant intercourse with the promoters of general science, escape the tendency to specialize their studies? How could medicine, as a part of science, remain conservative and sluggish in those times of minute observation and analysis, of subdivision of intellectual work, and of hungry original investigation?

3. The unexpected assistance afforded to medical research and practice by the progress of physics and the mechanical arts. No more striking example of an influence of this sort can be adduced than the effect of the introduction of the ophthalmoscope by Helmholtz in 1851. This instrument was not an accidental finding, but a truly scientific discovery resulting from the application of mathematics and physics to the study of the human eye. From this period dates the formal appearance of the first specialty, viz., ophthalmology: a specialty which has attracted to its study many of the brightest minds of our profession, which has accumulated discoveries upon discoveries, and, partly owing to its being largely founded upon exact sciences, has carried diagnosis to a remarkable degree of accuracy, and brought its various therapeutic measures to a rare degree of perfection.

The study of diseases of the cavities of the body, such as the nose, pharynx, larynx, and the more deeply placed organs has been greatly advanced by the invention of examining and illuminating apparatus.

The microscope has no doubt facilitated the growth of many a fine-spun and baseless theory, but it has certainly done much to enlarge the domain of science in the direction of physiology, diseases of the skin and kidneys, tumors, etc. At the present time, by its means important researches into the relation between microscopic germs and diseases are being carried on by numerous competent observers.

There are still other reasons, not perhaps scientific, why physicians have been led to limit their practice to certain branches. One is the great amount of time needed to carry out certain procedures of diagnosis and treatment, as for example in ophthalmic practice, in electro-therapeutics, hydro-therapeutics, etc. Again, in the last twenty-five years there has been a marked tendency to attempt the amelioration of chronic and so-called incurable diseases. These praiseworthy efforts need much thought and time, and can hardly be carried out by the busy general practitioner. Lastly, there is a strong popular demand for the services of specialists. Our patrons understand the advantages of concentrated study and large experience in limited fields of practice. The public seek special advice in the shape of consultations, or even place themselves in the hands of specialists for a time, without any disloyal intention toward their family physician, who is often a personal friend.

Specialties, in study and in practice, have been, I believe, of advantage to medical science.

By limiting their attention to specified branches of medicine, a considerable number of physicians have relieved themselves of the fatiguing cares and complex duties of general practice, and have thus obtained an amount of leisure time for study, and a tranquillity of mind favorable to original research.

In this way they have been able to make a critical examination of the writings of other observers in their own and in foreign lands, to make and record minute observations upon the living human being and upon the dead body, to undertake physiological experiments and anatomical researches intended to afford a logical basis for pathological hypotheses, and for an attempt at more rational therapy, and, finally, to accumulate experience in the comparatively rare diseases which general practitioners can only see at long intervals of time.

The results of these special studies, in a variety of departments, are beginning to take shape before us, as an unfinished yet a promising monument.

Each specialty can now point with pride to the numerous discoveries made by its followers; each can show a record of enthusiastic work, of keen discussions, of undoubted progress carried on or made public in its special organization or society.

The literature of each specialty has grown to be immense; embracing systematic works, pamphlets, and periodicals in many languages, and taxing to the utmost the industry of the specialist who means to be well-read in his branch of medicine. In this connection, I might incidentally remark that a knowledge of the three great living languages is now almost a *sine qua non* of success in special study.

The various specialties have, few will deny, proved useful to the public. I believe that multitudes of suffering human beings have been relieved or cured by specialists in the last thirty years, and that many, if not a majority, of these cases would not have been successfully treated by general practitioners, however learned and able they might have been. This proposition could be brilliantly supported from the records of ophthalmology, but every specialty can claim corresponding achievements.

By limiting his range of practice, a physician in the course of a few years accumulates a large experience in the diagnosis, prognosis and therapy of certain diseases, many of which are looked upon as quasi-incurable, and are almost shunned by the general practitioner.

Specialties are further useful to the public because they furnish peculiarly well qualified consulting physicians and surgeons. The willingness of general practitioners to seek special advice is becoming more and more evident. Even with our awkward rules of consultation, there need not be, I believe, any hostility or friction between the family physician and the specialist. The few unpleasant consultations of which I have been cognizant had been made so by personal faults in the physicians concerned.

I would venture to suggest that, on the one hand, the specialist who is saturated with the belief that he is the embodiment of science in his department, and who believes that the general practitioner cannot and does not know much in the same field, and, on the other hand, the general practitioner who is constitutionally unwilling or unable to have his diagnosis corrected or reversed, or to yield to the greater experience of the consulting physician—that both these men are equally ill-prepared for the delicate and important duties of consultation.

To these favorable comments I am compelled to add a few words of warning respecting the intellectual dangers which I believe attend special practice.

The first, or more evident, risk is that the specialist shall become a routine practitioner. This is, however, to a certain extent inevitable and justifiable. If, for example, a dermatologist find that a certain ointment is perfectly successful in the treatment of some diseases, who shall blame him if he continue to prescribe the same ointment in similar conditions of the skin? A given form of instrument is found best adapted to relieve a certain deformity or displacement: shall we apply the term routine practitioner, in any opprobrious sense, to the orthopedist who applies this instrument one hundred or more times a year?

By no means. This is a necessary routine, a useful routine, and one which it would be unwise to break through for the sake of sham originality.

But when such routine practice lulls a man asleep to the progress of his art, when it makes him blind and deaf to the improvements of others, when it prevents him from experimenting and trying to find something better, something which shall cure more quickly or with less pain or annoyance, then routine becomes a vice. Perhaps the mental state of the specialist who thus rests upon his oars, good oars though they be, might aptly be called one of partial dementia—a condition in which the past is remembered and overestimated, the living present is ignored, and the pregnant future unthought of.

A second danger in special practice is the tendency to acquire a belief in the specific potency of drugs, as contra-distinguished from their use in accordance with indications furnished by the patient's actual condition. As examples, I may quote the indiscriminate use of quinia in periodical symptoms, or the now fashionable prescription of a bromide for insomnia, or the application of electricity for paralysis. Do we always pause to consider that some remarkably periodical symptoms are not malarial, but of nervous origin—that insomnia is merely a symptom, which may depend upon various pathological conditions, and which is sometimes more quickly relieved by stimulants than by sedatives—or that many cases of paralysis get well spontaneously, or advance fatally, regardless of our electrical apparatus?

Closely attached to specific medication is the graver fault which I may term symptom-worship. Naturally enough, the specialist's attention is taken up with the very striking symptom which has caused the patient to consult him; such as convulsions, headache, eczema, failing sight, aphonia, etc. Some physicians, I fear, at once prescribe a favorite remedy or application in obedience to a half-avowed belief in specific medication. Others give more time to the case, analyze it somewhat, and prescribe intelligently. But how many have the courage to thoroughly investigate the problem, and then base their special practice in the case in hand upon the solid foundation of general medical knowledge? To do this consumes time, may call for delicate manipulations, and the acquired data must be submitted to a peculiar compound of inductive and deductive reasoning, in order to form a clear conception or hypothesis of the symptoms presented by the patient, as explained by general physiological, pathological and ætiological laws.

Without such an inquiry, how can we hope to construct a rational treatment?

Allow me to repeat that symptom worship and specific prescribing must flourish in due proportion to the neglect of general pathology by specialists. This idea is the substance of one of the chief arguments against the usefulness of specialties. It is claimed that the specialist is necessarily one-sided; that he carries on his researches and his practice in a mole-like way, *i. e.*, working in a furrow and ignoring its relations to the general system of medicine. I think that such a charge is unjust when applied in a general way; and I believe that, as years go on, fewer and fewer specialists will render themselves open to this serious accusation.

Having discussed the origin, utility, and dangers of specialties in medicine, there remain some practical deductions or advice to be addressed to you personally.

You will recollect that at the opening of my address I said that one of the important questions which agitate the minds of students and young graduates in medicine is, whether to become specialists or not.

Now, this question, like one or two others equally personal which will occupy your thoughts, I earnestly beg you not to be in any haste to decide. Pray do not, as the popular phrase is, "take up a specialty," for it seems to me that few things can be more unfortunate than that a young man, whether student or graduate, should label himself a specialist in his own mind or in the world's eye.

On the contrary, let your aim for several years be to cultivate your profession in a general way, with all the industry and the time which a providential lack of private patients will leave you This, I need hardly explain, is to be done by systematic reading and study, by hospital experience, and by the general practice of your art. For, even if you are ultimately to become specialists, let me assure you that you cannot be too well grounded in general diagnosis, in general therapeutics, and in anatomical and physiological knowledge.

During these years of preparatory study and work, not by any means unpleasant years to look back to, it may happen that you become greatly interested in some one branch of medicine, that circumstances lead you to see many diseases of a certain class; and that you experience a real desire, an ambitious desire, to cultivate this specialty. Thus, and then, if surrounding social conditions are favorable, if your medical friends consider that you are wise in your choice, a career as a specialist is open to you. This is what I would call a physician's natural growth into specialism.

In contra-distinction to the above rational process of first securing a thorough post-graduate medical education, and then carefully following one's intellectual bias in the choice of a special study or practice, I would hold up to you as a warning the course of those who, soon after graduating, with or without residence in a hospital, say to themselves, Let us be specialists—oculists, dermatologists, gynecologists, or what not. A certain fashion seems to determine which of the specialties is to be "taken up" by these hasty wooers. Some years ago ophthalmology was the proper thing; later still neurology was sought after; now I suspect (one can't be quite sure of contemporary movements) that gynecology is popular. I believe that such a course is a great evil for those who adopt it, for the unfortunate patients who fall in the hands of these pseudo-specialists, and finally harmful for the scientific reputation of other men who properly cultivate the special fields of medicine.

I have heard it whispered, pretty loud too, that some young men proclaim themselves specialists or "take up a specialty" under the delusion that a special practice is easy and very remunerative. Now, I am not disposed to deny that some few specialists are in the end handsomely rewarded; but who, save these favored few, realize what patient waiting, and what long-continued labor are implied by this success? Then, how many would-be specialists toil and wait, yet never come to be recognized as such by their *confrères* and by the public? There must be a mingling of remorse with great disappointment after having thus spent years in an artificial attempt to be a specialist without reward.

Yet, I do not wish to be understood as maintaining that the honest and well-prepared student of a specialty *must* succeed. No, gentlemen, there is no Sunday-school, good-boy doctrine in such real-life questions. The artificial, ill-grounded, relatively ignorant special practitioner may make money and even attain a certain distinction, while his neighbor, who has carefully and conscientiously worked his way along so as to be looked upon with respect by his associates, and even quoted as an authority, may fall short of success. This is because there enters into the problem of success in the practice of medicine a personal or social element of great importance, and which studious, original, and independent men are very apt to ignore. The successful physician is nearly always something more than learned: he is personally agreeable to his patients.

Finally, in considering whether you are to be specialists or not, I would have you bear in mind the normal organization of the great professional body which you join on graduating.

The immense majority of our brethren are settled in the country, in small villages and small cities, and they of necessity must be general practitioners. All honor to these men, forty thousand of them, I presume, who labor day and night, to the best of their ability and knowledge, for the relief of their neighbors' ailments. We can see them, with the help of our imagination, floundering through snow storms in the North, plodding along on horseback in the scorching sun-heat of the South, venturing into malarious regions, treating and even nursing contagious diseases, missing their meals—in fact, often shortening their lives to prescribe for the sick at rates of remuneration which we in New York consider ridiculously small. What matter if these men do not know all the fine points in medical science, if they have never heard of the depressor nerve, or do not know the name of the laryngeal muscles, or if they cannot establish the minute distinctions between various spinal paralyses? What if several times in their lives of usefulness, placed face to face with an unique or a complicated case, without the help of special counsel, they do too little or even do wrong? Will any one regret it more than they? And again, who, even in the elect circles of medical centres, does not also fail sometimes?

This great mass of the profession in the country and in cities, of which the majority of you must ultimately form a part, I greatly respect, all the more because I am the grandson of a physician who, for more than sixty years, was a useful and respected general practitioner in town and country.

Evidently, only a few physicians can, in obedience to the law of supply and demand, be specialists; and these few are found grouped according to certain geographical circumstances. No one would venture to attempt special practice in a village or in a large town. Even in cities of from fifty to one hundred thousand souls there is barely a living for one specialist in each department. Usually, special cases in such thinly populated regions are treated by one whom I may call, with no intended disrespect, the quasi-specialist. He is a general practitioner who has devoted time and pains to acquiring special knowledge and skill in the treatment of certain diseases. Other physicians are glad to send special practice to such a medical man if he be well qualified and honest in his professional relations; yet there may not be enough of such work to warrant his relinquishing general practice. Even in large cities there are many excellent physicians who might likewise be classed as quasi-specialists, yet I cannot but suspect that their special practice is dwindling as the public acquire more liking for strictly special advice and care.

There is, besides, a rather unclassified sort of physician in large cities, who is, to parody Molière, a specialist *malgré lui*; or, if you prefer it, a specialist *nolens volens*. This highly respectable gentleman usually expresses contempt for specialists; he looks upon them as narrow-minded, half-blind men working in a rut. He himself is widely read in medical lore, he may be a sort of walking encyclopedia, and he has practiced in all ways. Yet, fortunately for the public, though perhaps unfortunately for his grand ideal, this physician is known to his colleagues by his systematic work on this, or his lectures on that, or his monographs on various topics, etc. He is *de facto* a specialist; his *confrères* and people generally know that his opinion is particularly valuable in certain affections, and his consultation practice is colored accordingly. If he be a professor or a writer, his lectures and books reveal what really is in him in spite of a show of universal wisdom; and the bulk of what he writes is commonplace alongside of that part which treats of the topics he has unconsciously specialized.

Lastly, in all large cities there are the pure or strict specialists, that is, physicians who decline all practice outside of their specialty. I greatly hope to see this small class somewhat enlarged, mainly for the reason that we would then have a larger number of well-qualified observers with leisure to work, and thus a marked impetus would be given to original medical research in this country.

In this address I have endeavored to show that the growth of specialties has been normal, and in accord with the general scientific movement of the age; that specialties are useful to the public and to medical science; and that the practice of a specialty is not unattended by intellectual dangers.

I have neither urged you to become specialists nor advised you to shun and contemn specialties; but have tried to make plain and forcible my opinion, that the decision to become a specialist should be reached deliberately, upon a careful estimate of the tendencies and capabilities of your minds; and that your special studies and practice should rest upon a broad and solid medical culture.

Very few of you can expect to become strict specialists, and the career of all the rest as quasi-specialists and general practitioners will be equally useful, equally desirable socially, and equally honorable.

INDEX.

ABSCESS following injections of quinine, 22
Abscess of left frontal lobe of cerebrum, 452
Abuse and use of bromides, 226
Aconitia in the treatment of trigeminal neuralgia, 367
Aconitia, doses and formula. 368
do. in posterior spinal sclerosis, 492
Actual cautery, use of in medicine, 393
Acute central myelitis, case of, 412
do. ocular œdema, case of, 56
do. poliomyelitis in children, 84
Alkaline water as a vehicle for bromide of potassium, etc., 529
Aneurism of the cœliac axis, 639
Aphasia, history and pathology of, 27
Arsenical myelitis, 616
Arterial supply of the brain, 340
Ataxia, progressive locomotor, 353
do. as a symptom, 357
Autopsies in infantile paralysis, 95

Basal meningitis in children, 251
Basis cerebri, lesions of, 318
Birdsall's foot-dynamometer, 560
Bromide of potassium in hay-asthma, 241
Bromide of potassium in epilepsy, 234
Bromides, their abuse and use, 226
Bromism, 228
Bulbar paralysis, case of, 249

Cancer of vertebræ and paraplegia, 612
Cannabis indica for migraine, 242
Cautery, mode of application, 398
do. forms of, 395.
Cerebellum, diseases of, 335
Cerebral localizations, contributions to, 27, 202, 337, 407, 452, 495, 609, 647
Cerebral syphilis, a clinical lecture, 276
Cervical paraplegia, clinical study of, 256
Cervical sympathetic nerve, in a case of unilateral sweating of the head, 72
Cervico-brachial neuralgia and aneurism of the innominate artery, case of, 636
Coincidence of optic neuritis and subacute transverse myelitis, 421
Contracture in hemiplegia, physiology of, 132, 451
Cortex cerebri, physiology of, 337
do. do. lesions of, 337
Cortical centres, 341
Cranio-cerebral topography, 348, 647
Cultivation of specialties in medicine, 672
Cutaneous lesion from bromide of potassium, 629

Deformities in infantile paralysis, 87
Dermatitis produced by opium, 380
do. produced by bromide of potassium, 629
Description of a peculiar paraplegiform affection, 127
Desquamation of the kidneys during the administration of mercury and iodide of potassium, 403
Diagnosis of progressive locomotor ataxia, 353
Diagnosis of some organic diseases of the nervous system, 457
Diagnosis of paralytic dementia, 464
do. of tumor of the brain, 467
do. methods of, 558
Diagrams for cranio-cerebral topography, 350, 648, 649, 651
Diagram of motor cortical centres, 578
do. of spinal areas, 486
Diphtheritic ataxia, case of, 493
Disseminated cerebro-spinal sclerosis, cases of, 264
Dosage of fluid extract of conium, 596
do. of aconitia, 599
do. of phosphorus, 602
do. of nitrate of silver, 605
do. of bromides, 235
Double central canal of spinal cord, 74

Early diagnosis of organic nervous diseases, 457
Early recognition of epilepsy, 540
Efficient dosage of certain remedies used in the treatment of nervous diseases, 594
Ependyma, granulations of, 67
Epilepsy, treatment by bromides, 234
do. importance of its early diagnosis, 540

Epileptic and eclamptic attacks, 550
Excision of brachial plexus for neuralgia, 97

Folie à deux, 405
Formulas of bromide solutions, 235
Frontal lobe, abscess of, 452
Fulgurating pains, 355, 458, 463

General paresis of the insane, case of, 76
Glycosuria, cases of, 644
Granulations of cerebral ventricles 67

Headache and choked disc with tumor of the brain, 609
Hemianopsia, physiology of, 325
do. diagram of, 327
Hemiplegia, beginning in the foot, case of, 407
do. spinal, case of, 57
Hemorrhagic pachymeningitis, case of, 416
Hyoscyamia as an hypnotic, 472
do. in insanity, 477
do. as a depresso-motor, 484
do. paralysis agitans, 485
Hyoscyamia, formula for hypodermic use, 486
Hyoscyamia, in chorea, 488
Hypodermic injection of quinine, 22
Hysterical hemi-anæsthesia in a male subject, 641
Hysterical symptoms in organic nervous affections, 180

Infantile spinal paralysis, 84
Inhibitory arrest of the act of sneezing, 118
Injury to motor area of the brain, case of, 608
Insanity, contagion of, 405
Internal capsule, lesions of, 333
Intra-buccal method of faradizing the facial muscles, 419
Iodide of potassium in non-syphilitic organic nervous diseases, 579

Lectures on the localization of spinal and cerebral diseases, 283
Localization of spinal and cerebral diseases, lectures on, 283
Localization of diseases of the spinal cord, 436
Localized lesions of the cortex cerebri, 407
Localized basal meningitis in children, 251
Localized cerebral lesions, first contribution, 202 ; second contribution, 494

Malarial fevers, treated by subcutaneous use of quinine, 15
Mania, autopsy of case, 62
Medulla oblongata, lesions of, 316
do. do. structure of, 311
Medicinal treatment of chronic trigeminal neuralgia, 375
Melancholia, its treatment at home, 655
Mercury and iodide of potassium, effects on kidneys, 403
Metallo-therapy, case of, 641
Methods of diagnosis in nervous diseases, 558
Migraine, its treatment by Indian hemp, 242
Movable kidneys, case of, 415
Myelitis with optic neuritis, 421
do. following acute arsenical poisoning, 616
Mysophobia, case of, 429

Neuralgia, treatment by subcutaneous use of quinine, 10
Neuritis, pathological anatomy of, 106
do. of traumatic origin, 401

Occipital headache as a symptom of uræmia, 432
Opium, eruptions from, 380
Optic neuritis and myelitis, 421
Outline of the physiology of the nervous system, 164

Pachymeningitis, cases of, 195
Paraplegia in syphilitic subjects, 413
Pathology of infantile paralysis, 90
Pedal neuralgia, traumatic, 401
Physiology of the nervous system, 164
Pneumonia, temperature of, 1
Poliomyelitis, after injury, 381
do. infantile, 84
Post-paralytic chorea, clinical study of, 197
Pyramidal decussation, 312, 450

Quinine subcutaneously in malarial neuralgia, 10
do. in malarial fevers, 15

Simulated glycosuria, 645
Slow pulse and epileptiform convulsions, case of, 409
Sneezing, historical and physiological considerations, 118
Solution of quinine for hypodermic use, 15
Solution of hyoscyamia, 486
Specialties in medicine, 672
Speech, mechanism of, 33
Spider cells in sclerosis, 270
Spinal cord, localized diseases of, 436
do. lesion of one-half of, 57
do. focal lesions of, 304
do. systematic lesions of, 293
do. columns of, 289

Spinal hemiplegia, case of, 57
do. physiology, 309
do. paralysis in children, 84
Statement of the aphasia question, 27
Subcutaneous use of quinine in malarial neuralgia, 10
Subcutaneous use of quinine in malarial fevers, 15
Syphilitic paraplegia, 418

Tabes dorsalis, differential diagnosis, 364
Table of hysterical symptoms, 181
Tetanoid paraplegia, 127, 383
Theory of cerebral contracture, 132
Therapeutic contributions, 10, 15, 97, 139, 226, 242, 256, 367, 375, 393, 401, 419, 472, 484, 492, 529, 534, 579, 594, 641, 655
Therapeutics of the nervous system, 138
Thermometer in clinical medicine, 1
Tracings of slow pulse, 410
" of paralysis agitans, 485
Traumatic brachial neuralgia, case of, 97
Traumatic epilepsy, case of, 608
do. pedal neuralgia, case of, 401
do. poliomyelitis, case of, 381
Trichinosis, case of, 55
Trigeminal neuralgia, treatment of, 367, 375, 534
Treatment of cervical paraplegia, 256
do. of migraine, 242
do. of trigeminal neuralgia, 367

Uræmia, with occipital headache, 432
Ulcus elevatum from use of bromides, 629

Van der kolk's law in diagnosis, 574
Vertebral cancer and paraplegia, case of, 612

Word-deafness, case of, 508

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